TO EVALUATE THE ROLE OF BLOOD MARKERS (HAEMATOLOGICAL AND BIOCHEMICAL) AS PREDICTORS FOR DEVELOPMENT OF PRESSURE SORES

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Dedicated

to

my family

&

Teachers

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Dr. Deepika Sharma

LIST OF ABBREVIATIONS

ICU	- Intensive care unit
BC	- Before Christ
NPUAP	- National Pressure Injury Advisory Panel.
EPUAP	- European Pressure Ulcer Advisory Panel
ICD11	-International Classification of diseases
WHO	-World health organisation
DKA	-Diabetic ketoacidosis
LRTI	- Lower respiratory tract infection
CKD	- Chronic kidney disease
DM	- Diabetes mellitus
HTN	- Hypertension
CAD	- Coronary artery disease
COPD	- Chronic Obstructive Pulmonary Disease
CLD	- Chronic Liver disease
HCV	- Hepatitis C Virus
HIV	- Human Immunodeficiency Virus
PHTN	- Portal Hypertension
Hb	- Hemoglobin
P value	- Probability value
SD	- Standard Deviation
IQR	- Interquartile Range

ABSTRACT

Introduction: Pressure sores are an important public health problem associated with increased morbidity, reduced health- related quality of life, prolonged hospital length of stay, and high treatment costs. The Braden scale is one of the most widely used risk assessment scale for them with high negative predictive values. Present study is aimed to study haematological and biochemical parameters like haemoglobin, platelet count, serum albumin, direct and indirect bilirubin, and creatinine which can be considered for the risk evaluation for the pressure sore occurrence.

Aim of Study: To evaluate the role of blood markers (haematological and biochemical) as predictors for development of pressure sores.

Materials & Methods: This will be a prospective study in patients admitted to Medicine ICU whose Braden Assessment Scale will be done and patients with score less than 18 will be included in the study. Patients will be divided into two groups; Group A(patients without pressure sores and Group B(patients with pressure sores). Derranged value of recorded haematological and biochemical markers will be compared in both the groups using appropriate statistical methods.

RESULTS: The distribution of pressure sores across age groups shows that the highest occurrence is in the 51-80 age group, with no significant association between age and pressure sores (p-value = 0.377). Similarly, there is no statistically significant association between sex and pressure sores (p-value = 0.437), though males had a slightly higher incidence than females. However, significant associations were found between pressure sores and the Braden scale (p-value = 0.009), hemoglobin levels (p-value = 0.001), platelet counts (p-value = 0.005), creatinine levels (p-value = 0.019), and bilirubin levels (p-value = 0.002 for indirect bilirubin). Lower hemoglobin, platelet, and albumin levels, along with higher creatinine and bilirubin levels, were linked to a greater risk of developing pressure sores.

DISCUSSION: This study found that males and older adults are more susceptible to pressure sores, particularly in the sacral area. Lower hemoglobin and platelet levels, as well as higher indirect bilirubin and creatinine levels, were significantly associated with pressure sore development, while albumin levels showed no strong correlation.

Conclusion: This study highlights the importance of integrating hematological and biochemical markers with existing tools like the Braden Scale for improved prediction of pressure sore development. Significant correlations were identified between lower hemoglobin and platelet levels, as well as elevated indirect bilirubin and creatinine levels, and the occurrence of pressure sores, suggesting these markers can enhance clinical risk assessment.

Keywords: Braden Assessment Scale, Pressure Sore, Haematological and Biochemical Markers.

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INTRODUCTION

Pressure sores, often called bedsores, decubitus ulcers, or pressure ulcers[1], develop when sustained pressure impairs blood flow to specific areas of the skin and underlying tissues, typically over areas where bones are close to the skin surface like occiput, scapula, sacrum, buttocks, ischium, heels, and toes. This pressure reduces circulation and oxygen to the tissues, leading to cell death and ulceration [1]. Sacrum, ischial tuberosity, and greater trochanter are prominent sites due to their bony prominence and the fact that these areas often endure prolonged pressure, especially in individuals who are bedridden or use a wheelchair [2].

Pressure sores have been described extensively in the literature since the 19th century. Jean-Martin Charcot was a prominent French physician in the 19th century who made significant contributions to the understanding of various diseases, including pressure sores. He observed that patients who developed severe pressure sores, particularly on the buttocks and sacrum, often experienced poor outcomes and sometimes died as a result. Charcot referred to these lesions as "decubitus ominous," indicating that the presence of such ulcers was a serious and often fatal complication. His observations underscored the severity of pressure sores and their association with overall patient mortality [3].

Pressure sores are often described as having an "iceberg-like" nature, with the visible skin damage representing only the tip of the iceberg. Histologically, deep tissue injury is typically much more severe, even when the dermis remains intact in the early stages of ulcer development.

There are several factors contributing to the development of pressure sores include age, dehydration, malnutrition, incontinence, friction, and shear forces [4]. A high incidence of pressure sores is considered a negative outcome of care, significantly impacting patients' quality of life, morbidity, and mortality [5]. Pressure sore occurs in very ill patients and who are kept in prolonged immobilisation. They are quiet frequent in

intensive care units and in paraplegic individuals. These patients are at a particularly high risk of developing pressure sores due to sedation, mechanical ventilation, and prolonged bed confinement, which significantly increase the risk of skin breakdown [6,7].

The development of pressure sores is linked to several negative effects on patients, including pain, depression, loss of function and independence, increased risk of infection and sepsis, and the need for additional surgical procedures, all of which contribute to a longer hospital stay. The most vulnerable groups include: 1) patients with spinal cord injuries; 2) elderly individuals; 3) hospitalized patients, particularly those who have undergone orthopaedic surgery; and 4) patients admitted to the intensive care unit (ICU). The last group, in particular, is at a high risk of developing pressure sores due to prolonged immobility [8].

The development of pressure sores in critically ill patients is a preventable complication [9]. As a result, several pressure sores risk assessment tools, such as the Braden, Waterlow, Norton, and Cubbin–Jackson scales, have been developed [10].

The Braden Assessment Scale is the most widely used tool for predicting the risk of pressure sore development. It evaluates six key factors that contribute to pressure sore formation: mobility, activity, sensory perception, skin moisture, nutritional status, and friction/shear. Each factor is rated on a scale from 1 to 4 (with friction/shear rated from 1 to 3), where a lower score indicates a higher risk. The total score ranges from 6 to 23, with scores below 18 commonly used in clinical practice to identify at-risk patients. However, the Braden Scale has demonstrated low specificity and positive predictive value, resulting in a high rate of false positives. This overprediction can lead to unnecessary and costly interventions that may not be the most efficient use of healthcare resources. As a result, there is a need for more accurate methods to predict pressure sore development, particularly in ICU settings [11].

There is an established relationship between nutritional status and the development of pressure sores. While treatments like mechanical ventilation and medications are crucial for survival, preventing malnutrition is equally important. Poor nutrition weakens the body and greatly increases the risk of pressure sores, which develop when the skin and underlying tissues are damaged from prolonged immobility.

Presence of critical illness triggers stress responses that lead to rapid muscle loss, further contributing to weakness and weight loss. This makes patients more susceptible to pressure sores. It has been observed that many of these patients have low levels of albumin, which can be monitored as a biomarker to predict the likelihood of developing pressure sores [12].

In addition to albumin, other biochemical markers such as bilirubin and creatinine provide insight into liver and kidney function. In chronically ill patients, who are at higher risk of multiple organ dysfunction syndrome, these laboratory parameters often become abnormal. Recent studies have shown that patients with deranged liver and kidney markers are more prone to pressure sores, highlighting the potential of these biomarkers in the early prediction and prevention of this serious complication.

Monitoring and optimizing nutrition, based on changes in body composition and biochemical parameters, are key factors in reducing the risk of pressure sores. Implementing reliable assessment method along with biochemical, and haematological parameters, may result in predicting pressure sore in critically ill patients [12].

The role of haematological and biochemical markers in predicting the development of pressure sores has been examined in various studies individually. However, to the best of our knowledge, no single study has combined these markers to draw comprehensive conclusions. This is the first study to integrate both haematological markers, such as hemoglobin and platelets, and biochemical markers, including albumin, bilirubin, and creatinine, in assessing pressure sore risk. Since these laboratory tests are routinely conducted during hospital admissions,

they provide an accessible and cost-effective method for monitoring and predicting the risk of pressure sores [12].

The present study is designed to evaluate the role of haematological as well as biochemical blood markers in predicting the development of pressure sores.

AIM AND OBJECTIVE

To evaluate the role of blood markers (haematological and biochemical) as predictors for development of pressure sores.

REVIEW OF LITERATURE

Pressure sores have been recognized as a medical condition for thousands of years. Evidence of pressure sores has been found in Egyptian mummies over 5,000 years old, with honey used as a treatment for such ulcers and wounds. In Persia, Avicenna applied various topical treatments for wounds, while in Arabia, Maimonides emphasized nutritional support to aid ulcer healing. Historically, a wide range of topical remedies, such as honey, moldy bread, meat, plant and animal extracts, copper sulfate, zinc oxide, and alum, were used[13].

Hippocrates (460-370 B.C.) described pressure ulcers in relation to paraplegia, bladder, and bowel dysfunction[14]. During the Renaissance, Ambrose Paré, a 16th-century French army surgeon and a pioneer of modern surgical practice, wrote about a wounded aristocrat developing a pressure sore, which was treated with nutrition, pain relief, and debridement—methods still used today to some extent [15].

In the 19th century, Jean-Martin Charcot studied decubitus ulcers, proposing the "neurotrophic theory" of ulcer formation, linking it to central nervous system damage rather than solely to pressure. Charcot coined the term "decubitus ominosus" for ulcers covered with eschar, which were often fatal. He described complications like gangrenous pulmonary metastasis and spinal cord invasion. However, Brown-Séquard opposed Charcot's theory, demonstrating in spinal cord-injured guinea pigs that sores could be prevented and healed by relieving pressure [3].

In Ayurveda, pressure sores are referred to as "Sayyaj Vrava," where "Sayyaj" means bed and "vrava" means ulcer or wound. The interruption of circulation ("mamsadhatu") due to pressure is believed to cause bedsores.

The 19th century saw significant advancements in understanding ulcers with Pasteur's discovery of bacteria, Lister's antiseptic methods, and Roentgen's X-ray. The 20th century brought antibiotics, further

revolutionizing treatment, while later research focused on nutrition, trace elements, biomechanics, and new management strategies for treating pressure sores [16].

EPIDEMIOLOGY

Pressure sores incidence rates vary significantly across healthcare settings. In the United States, up to 3 million adults are affected with pressure sores annually[2]. Managing pressure sores represents a significant economic burden. In 2019, Padula and Delarmente estimated that the cost of hospital-acquired pressure sores could exceed $26.8 billion. Their study simulated an incidence of 8.3 hospital acquired pressure sores cases per 100 acutely ill patients, aligning with earlier research indicating that 7.9% of at-risk patients develop pressure sores after getting admitted in hospitals [17].

Sacral decubitus ulcers are particularly common among elderly patients. Those who are incontinent, paralyzed, or otherwise debilitated are at a higher risk. Patients with normal sensory function, mobility, and cognitive status are less likely to develop these ulcers, as their physiological feedback mechanisms lead to regular changes in position. As noted, older adults are more susceptible, with two-thirds of ulcers occurring in patients over 70. Additionally, data shows that 83% of hospitalized patients develop pressure sores within the first five days of hospitalization [17].

NOMENCLATURE

In 2016, the National Pressure Ulcer Advisory Panel (NPUAP) updated the terminology from "pressure sore" to "pressure injury" to better reflect the underlying tissue damage that occurs before visible skin breakdown. While pressure sore implies a break in the skin, "injury" refers to the damage that can occur even when the skin remains intact.

The classification of pressure sores has evolved since 1955, with early systems focusing on the stages of tissue damage. The current system uses Arabic numerals to reduce confusion with medical terms like "IV"

(intravenous), and it emphasizes that pressure sores do not necessarily progress in a linear fashion from Stage 1 to Stage 4 [18].
Numerous classifications have been described for pressure sores. The National Pressure Ulcer Advisory Panel (NPUAP) has updated these classifications to reflect the latest understanding of the etiology of pressure sores. In 2018, the World Health Organization (WHO) introduced the ICD-11 [19], which includes a six-category classification for pressure ulcers/injuries, as outlined below:

CLASSIFICATION

- **Stage I**: Intact skin with non-blanchable erythema in a localized area, typically over a bony prominence.
- **Stage II**: Partial thickness loss of the dermis, appearing as a shallow open wound with a red or pink wound bed, without slough. It may also appear as an intact or ruptured serum-filled or blood-filled blister.
- **Stage III**: Full-thickness skin loss where slough may be present but does not obscure the depth of tissue loss. The wound may include undermining and tunnelling.
- **Stage IV**: Full-thickness tissue loss with exposed bone, tendon, or muscle. Slough or eschar may be present, often accompanied by undermining and tunnelling. Exposed bone or muscle may be visible or palpable.
- **Unstageable**: Full-thickness skin or tissue loss with an unknown depth. The wound bed is obscured by slough (yellow, tan, gray, green, or brown) and/or eschar (tan, brown, or black). The true depth cannot be determined until the slough or eschar is removed, but the wound is likely to be either Stage III or IV.
- **Suspected Deep Tissue Injury**: Depth unknown. A localized area of discolored, intact skin, either purple or maroon, or a blood-filled blister, resulting from damage to underlying soft tissue caused by pressure and/or shear.

ETIOLOGY

The development of pressure sores is a complex process influenced by multiple factors. Both external and internal factors contribute to its formation. Externally, prolonged pressure, friction, shear forces, and moisture can lead to tissue deformation and reduced blood flow (ischemia). Internally, conditions like malnutrition, anemia, and endothelial dysfunction accelerate tissue damage. The most common risk factors for pressure sores include decreased mobility, skin moisture, poor nutrition, and loss of sensory perception. In addition, older age, cognitive impairment, and comorbidities that hinder tissue healing have been identified as significant contributors [20].

Prolonged pressure on the skin can block blood flow to capillaries, reducing oxygen supply to tissues. Over time, this leads to the buildup of toxic waste products in the ischemic tissue, eventually causing tissue ulceration and necrosis. In bedridden or surgical patients, immobility for just two hours can be enough to trigger the development of a pressure sores. Impaired nervous system regulation of local blood flow also plays a role in pressure sores formation. Patients with the following conditions are at higher risk of developing pressure sores:

- Neurologic diseases
- Cardiovascular diseases
- Prolonged anesthesia
- Dehydration
- Malnutrition
- Hypotension
- Recent surgery

As the new definition indicates, prolonged pressure is the primary factor in pressure sore development. There is an inverse relationship between the intensity and duration of pressure: higher pressure over a short time can cause tissue necrosis faster, while lower pressure requires a longer duration to lead to tissue damage and ulceration.

High pressure applied for a short time not only causes tissue necrosis by blocking capillaries but also affects larger vessels, often leading to venous thrombosis. Therefore, the harmful effects of short-term high

pressure are greater than those of long-term low pressure. Studies have shown that even after high pressure is relieved, ischemia may persist due to damage to larger vessels. In contrast, after low pressure is relieved, a normal hyperemic response typically compensates for the temporary ischemia, preventing tissue degeneration [20].

PATHOPHYSIOLOGY

Pressure sore formation is a multifactorial process that ultimately leads to ischemia and necrosis. While tissues can tolerate abnormal external pressure to some extent, prolonged pressure is the primary cause. For blood flow to be obstructed, external pressure must exceed the arterial capillary pressure of 32 mm Hg, and for venous blood return to be impaired, it must surpass the venous capillary closing pressure of 8 to 12 mm Hg. Prolonged pressure above these thresholds results in tissue ischemia and necrosis, often caused by compression from firm mattresses, hospital bed railings, or other hard surfaces.

The European Pressure Ulcer Advisory Panel (EPUAP) also highlighted the connection between COVID-19 pathophysiology and the development of pressure sores. The panel identified pro-inflammatory cytokines such as interleukin-6 and tumor necrosis factor-alpha, commonly elevated in COVID-19 patients, as contributors to sustained inflammation following cell death, promoting pressure ulcer formation. Additionally, friction from skin rubbing against surfaces like clothing or bedding can damage the skin's superficial layers, while moisture exacerbates tissue breakdown and maceration, further increasing the risk of pressure sores [21].

ASSESSMENT TOOLS

The use of pressure sore risk assessment tools or scales is a crucial part of the assessment process for identifying individuals at risk of developing pressure sores. Notable examples of such risk assessment tools include the Norton Scale (Norton 1975), the Waterlow Scale (Waterlow 1985), and the Braden Scale (Braden 1987).

Nearly 40 different risk assessment scales are currently in use, many of which are derived from the foundational work of Norton (1975) or have been developed in response to literature reviews.

It is evident that risk factors for developing pressure sores can vary significantly among patients in different clinical settings. Consequently, it may be challenging to create a single risk assessment tool that is universally applicable across all patient populations and clinical environments [21].

The Braden Scale for predicting pressure ulcer risk is among the most widely utilized tools for assessing the likelihood of pressure sore development. It is one of only two pressure sore prediction tools recognized by the U.S. Agency for Health Care Policy and Research, with the other being the Norton Scale (U.S. Department of Health and Human Services, 1992)

Braden Assessment Scale is most frequently used scale for predicting pressure sore occurrence. It was developed in 1984 by Barbara J. Braden and was published in 1987. The Braden scale evaluates the risk of skin breakdown in six domains: sensory perception, moisture, activity, mobility, nutrition, and friction and shear [22].
It is given below:

BRADEN SCALE – For Predicting Pressure Sore Risk

SEVERE RISK: Total score ≤ 9 **HIGH RISK**: Total score 10-12 **MODERATE RISK**: Total score 13-14 **MILD RISK**: Total score 15-18				DATE OF ASSESS ➡				
RISK FACTOR	**SCORE/DESCRIPTION**				**1**	**2**	**3**	**4**
SENSORY PERCEPTION Ability to respond meaningfully to pressure-related discomfort	**1. COMPLETELY LIMITED** – Unresponsive (does not moan, flinch, or grasp) to painful stimuli, due to diminished level of consciousness or sedation, **OR** limited ability to feel pain over most of body surface.	**2. VERY LIMITED** – Responds only to painful stimuli. Cannot communicate discomfort except by moaning or restlessness, **OR** has a sensory impairment which limits the ability to feel pain or discomfort over ½ of body.	**3. SLIGHTLY LIMITED** – Responds to verbal commands but cannot always communicate discomfort or need to be turned, **OR** has some sensory impairment which limits ability to feel pain or discomfort in 1 or 2 extremities.	4. **NO IMPAIRMENT** – Responds to verbal commands. Has no sensory deficit which would limit ability to feel or voice pain or discomfort.				
MOISTURE Degree to which skin is exposed to moisture	1. **CONSTANTLY MOIST**– Skin is kept moist almost constantly by perspiration, urine, etc. Dampness is detected every time patient is moved or turned.	2. **OFTEN MOIST** – Skin is often but not always moist. Linen must be changed at least once a shift.	3. **OCCASIONALLY MOIST** – Skin is occasionally moist, requiring an extra linen change approximately once a day.	4. **RARELY MOIST** – Skin is usually dry; linen only requires changing at routine intervals.				
ACTIVITY Degree of physical activity	1. **BEDFAST** – Confined to bed.	2. **CHAIRFAST** – Ability to walk severely limited or nonexistent. Cannot bear own weight and/or must be assisted into chair or wheelchair.	3. **WALKS OCCASIONALLY** – Walks occasionally during day, but for very short distances, with or without assistance. Spends majority of each shift in bed or chair.	4. **WALKS FREQUENTLY**– Walks outside the room at least twice a day and inside room at least once every 2 hours during waking hours.				
MOBILITY Ability to change and control body position	1. **COMPLETELY IMMOBILE** – Does not make even slight changes in body or extremity position without assistance.	2. **VERY LIMITED** – Makes occasional slight changes in body or extremity position but unable to make frequent or significant changes independently.	3. **SLIGHTLY LIMITED** – Makes frequent though slight changes in body or extremity position independently.	4. **NO LIMITATIONS** – Makes major and frequent changes in position without assistance.				
NUTRITION Usual food intake pattern [1]NPO: Nothing by mouth. [2]IV: Intravenously. [3]TPN: Total parenteral nutrition.	**1. VERY POOR** – Never eats a complete meal. Rarely eats more than 1/3 of any food offered. Eats 2 servings or less of protein (meat or dairy products) per day. Takes fluids poorly. Does not take a liquid dietary supplement, **OR** is NPO[1] and/or maintained on clear liquids or IV[2] for more than 5 days.	**2. PROBABLY INADEQUATE** – Rarely eats a complete meal and generally eats only about ½ of any food offered. Protein intake includes only 3 servings of meat or dairy products per day. Occasionally will take a dietary supplement **OR** receives less than optimum amount of liquid diet or tube feeding.	**3. ADEQUATE** – Eats over half of most meals. Eats a total of 4 servings of protein (meat, dairy products) each day. Occasionally refuses a meal, but will usually take a supplement if offered, **OR** is on a tube feeding or TPN[3] regimen, which probably meets most of nutritional needs.	4. **EXCELLENT** – Eats most of every meal. Never refuses a meal. Usually eats a total of 4 or more servings of meat and dairy products. Occasionally eats between meals. Does not require supplementation.				
FRICTION AND SHEAR	1. **PROBLEM**- Requires moderate to maximum assistance in moving. Complete lifting without sliding against sheets is impossible. Frequently slides down in bed or chair, requiring frequent repositioning with maximum assistance. Spasticity, contractures, or agitation leads to almost constant friction.	2. **POTENTIAL PROBLEM**– Moves feebly or requires minimum assistance. During a move, skin probably slides to some extent against sheets, chair, restraints, or other devices. Maintains relatively good position in chair or bed most of the time but occasionally slides down.	3. **NO APPARENT PROBLEM** – Moves in bed and in chair independently and has sufficient muscle strength to lift up completely during move. Maintains good position in bed or chair at all times.					
TOTAL SCORE	**Total score of 12 or less represents HIGH RISK**							

ASSESS	DATE	EVALUATOR SIGNATURE/TITLE	ASSESS.	DATE	EVALUATOR SIGNATURE/TITLE
1	/ /		3	/ /	
2	/ /		4	/ /	

Each sub scale is ranked on a scale of 1 to 4, except for the friction/shear sub scale, which is scored in a scale of 1 to 3. Sub scores are added to determine the cumulative score that varies from 6 to 23; lower scores indicate higher risk for pressure ulcer development. A cutoff score of 18 or less indicates an increased risk for pressure ulcer development [22].

The Braden Scale is widely used due to its simplicity and ease of scoring, as frequently noted in the literature. However, multiple studies

suggest that it tends to over predict the risk of pressure ulcer development. Recent research has also evaluated its sensitivity and specificity, highlighting limitations in its predictive accuracy [23].

In addition to the above, there are other haematological as well as biochemical parameters like haemoglobin, platelet count, serum albumin, direct and indirect bilirubin, and creatinine that can be considered for the risk evaluation for the development of pressure sores.

The role of haematological and biochemical markers in predicting the development of pressure sores has been examined in various studies individually [24-27]. However, to the best of our knowledge, no single study has collaborated these markers to draw comprehensive conclusions. This is the first study to integrate both haematological markers, such as hemoglobin and platelets, and biochemical markers, including albumin, bilirubin, and creatinine, in predicting the development of pressure sores. Since these laboratory tests are routinely conducted during hospital admissions, they provide an accessible and cost-effective method for monitoring and predicting the risk of pressure sores.

Furthermore, it is known that low levels of hemoglobin and platelets are related to the pathological condition of anemia, which involves a lower transport of nutrients and oxygen to the tissues, and they are responsible for breaking skin integrity [28].

Many studies have been conducted to predict the role of biochemical markers like albumin, direct and indirect bilirubin and creatinine in prediction of pressure sores.

Patients admitted to ICU settings are often chronically ill and frequently exhibit low albumin levels, which is indicative of malnutrition. Studies have shown that these reduced albumin levels in chronically ill patients are often due to poor nutritional intake. Malnutrition among hospitalized patients remains a widespread issue, with an estimated 35–55% of patients affected, and around 20% requiring immediate nutritional intervention. Regular monitoring of key biochemical markers and continuous risk assessment for pressure sores using recommended

scales can significantly lower the risk of pressure sores and related complications. Implementing personalized nutritional care helps prevent the negative effects of malnutrition, and early detection should be a priority for medical staff [28].

In the tests conducted, only 10% of patients had normal albumin levels, despite experiencing body weight loss. Importantly, the patients with the lowest albumin values also had the highest CRP levels, suggesting that albumin may be mobilized during the production of acute-phase proteins. Serum albumin is considered a marker of both inflammation and cachexia, or possibly malnutrition. However, since dietary restrictions do not always lead to a decrease in serum albumin, it has been proposed that inflammation plays a more significant role than malnutrition in reducing albumin levels [27].

Research by Serra et al. on critically ill ICU patients found that lower albumin concentrations were associated with a higher incidence of pressure sores. Maintaining albumin levels at 2.8 g/dL or higher, along with protein supplementation, had a positive effect on the healing of pressure sores [29]. Similarly, Montalcini et al. reported that serum albumin levels below 3.1 g/dL were predictive of pressure sore occurrence and were linked to increased mortality rates [27].

The present study is aimed to compare the efficacy of braden assessment scale and various haematological and biochemical markers in prediction of pressure sore occurrence.

MATERIAL AND METHODS

Source of data

This prospective study was conducted on patients admitted in Medicine ICU at Dayanand Medical College and Hospital, Ludhiana from October 2022 to february 2024.

Method of collection of data:-

A) Inclusion criteria

- Patients admitted to Medicine ICU.
- Age more than 18 years.
- Braden scale less than 18.
- Expected ICU stay of 5 days or more.

B) Exclusion criteria

- Patients with pre-existing pressure sore.
- Patients not willing to participate in the study.
- Expected ICU stay of less than 5 days.

Study design

- This was a prospective study on patients being admitted to Medicine ICU.
- Informed consent was taken from the patient/family before including the patient in the present study.
- All the patients underwent detailed assessment within 12 hours of admission in ICU in the form of history and clinical examination of the skin over all bony prominences.
- Braden Assessment Scale was used daily to identify the patients who are at risk for pressure sore development.

BRADEN SCALE – For Predicting Pressure Sore Risk

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1	/ /		3	/ /	
2	/ /		4	/ /	

- Patients with Braden scale less than 18 were included in this study.
- Haematological and biochemical parameters like haemoglobin, platelet count, serum albumin, Direct and indirect bilirubin, and creatinine were recorded for these patients **biweekly** until their discharge.
- Head-to-toe skin examination over bony prominences was done to document the presence and stage of pressure sore.

- Patients were divided into two groups: - Group A -patients without pressure sores.Group B- patients with pressure sores.
  Derranged value of recorded haematological and biochemical markers were compared in both the groups using appropriate statistical methods.

OBSERVATION AND RESULTS

TABLE 1: AGE DISTRIBUTION

Age group(years)	No. of cases	Percentage
< 40	12	6.6%
41-50	27	14.8%
51-60	39	21.4%
61-70	54	29.7%
71-80	40	22.0%
> 80	10	5.5%
Total	182	100.0%

- In total, there were 182 patients across all age groups.
- There were 12 patients in <40 age group, 27 patients in 41-50 age group, 39 patients in 51-60 age group, 54 patients in 61-70 age group, 40 patients in 71-80 age group and 10 patients in >80 age group.
- The highest numbers of patients were in the 61-70 age group (54 patients, 29.7%).
- The lowest number of patients were in the >80 age group (10 patients, 5.5%).

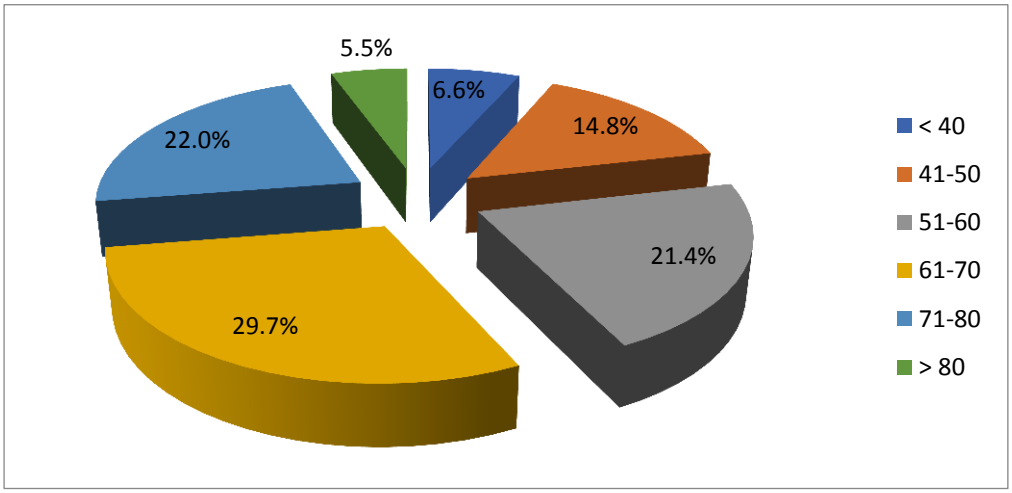

FIGURE 1: AGE DISTRIBUTION

TABLE 2: SEX DISTRIBUTION

Sex	No. of cases	Percentage
Female	71	39.0%
Male	111	61.0%
Total	182	100.0%

- Out of the total 182 cases, there were 71 females (39.0%) and 111 males (61.0%).

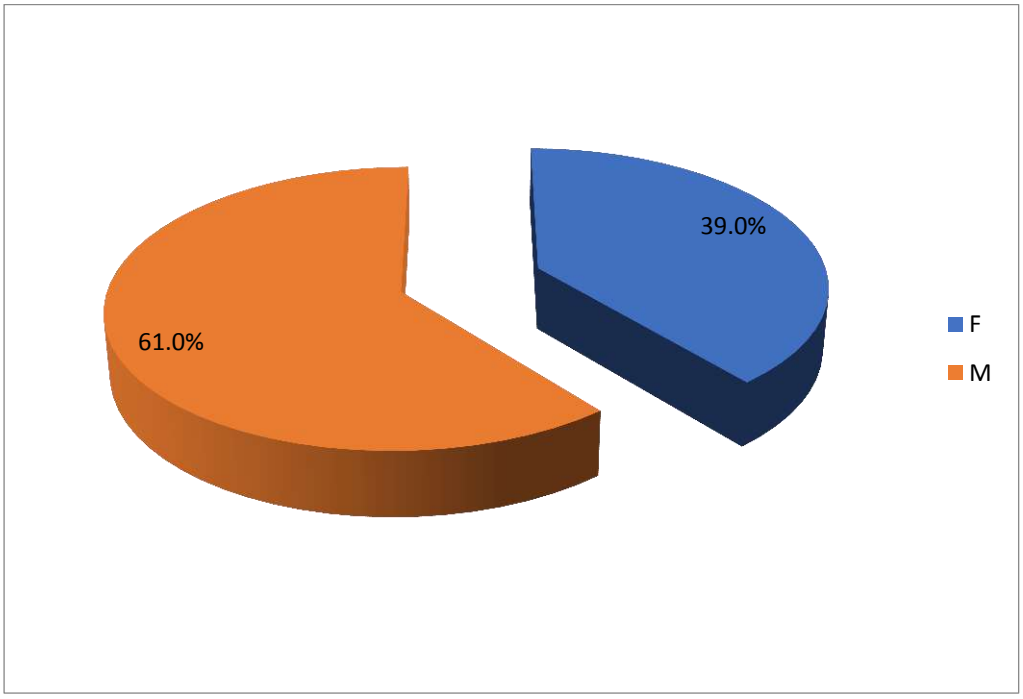

FIGURE 2: SEX DISTRIBUTION

TABLE 3: DISTRIBUTION OF PATIENTS ACCORDING TO DIAGNOSIS

Diagnosis	No. of cases	Percentage
Acute exacerbation of COPD	26	14.3%
Acute febrile illness	5	2.7%
Acute kidney disease	3	1.6%
Acute pancreatitis	4	2.2%
Acute poisoning	4	2.2%
Acute tubercular myelitis	1	0.5%
Acute viral hepatitis	2	1.1%
Chronic kidney disease	20	11.0%
Chronic liver disease	4	2.2%
Coronary artery disease	4	2.2%
DKA	29	15.9%
Guillain barre syndrome	2	1.1%
Hypertensive emergency	3	1.6%
Hypoxic brain injury	2	1.1%
Ischaemic stroke	9	4.9%
LRTI	11	6.0%
Metabolic seizures	8	4.4%
Septicemia	28	15.4%
Tubercular meningitis	1	0.5%
Urosepsis	16	8.8%
Total	182	100.0%

- The most common diagnosis was Diabetic Ketoacidosis (DKA), accounting for 29 patients (15.9%), followed by Septicemia in 28 patients (15.4%), and Acute Exacerbation of COPD in 26 patients (14.3%).

- Chronic Kidney Disease was diagnosed in 20 patients (11.0%), while Urosepsis in 16 patients (8.8%), and Lower Respiratory Tract Infection (LRTI) in 11 patients (6.0%).
- Other conditions included Ischemic Stroke, in 9 patients (4.9%), and Metabolic Seizures, contributing to 8 cases (4.4%).
- Acute Febrile Illness, Acute Kidney Disease, Hypertensive Emergency, Guillain-Barre Syndrome, Hypoxic Brain Injury, and Acute Viral Hepatitis each was diagnosed in 1-2% of the total patients.
- Acute Tubercular Myelitis and Tubercular Meningitis was seen in 1 patient (0.5%) each.

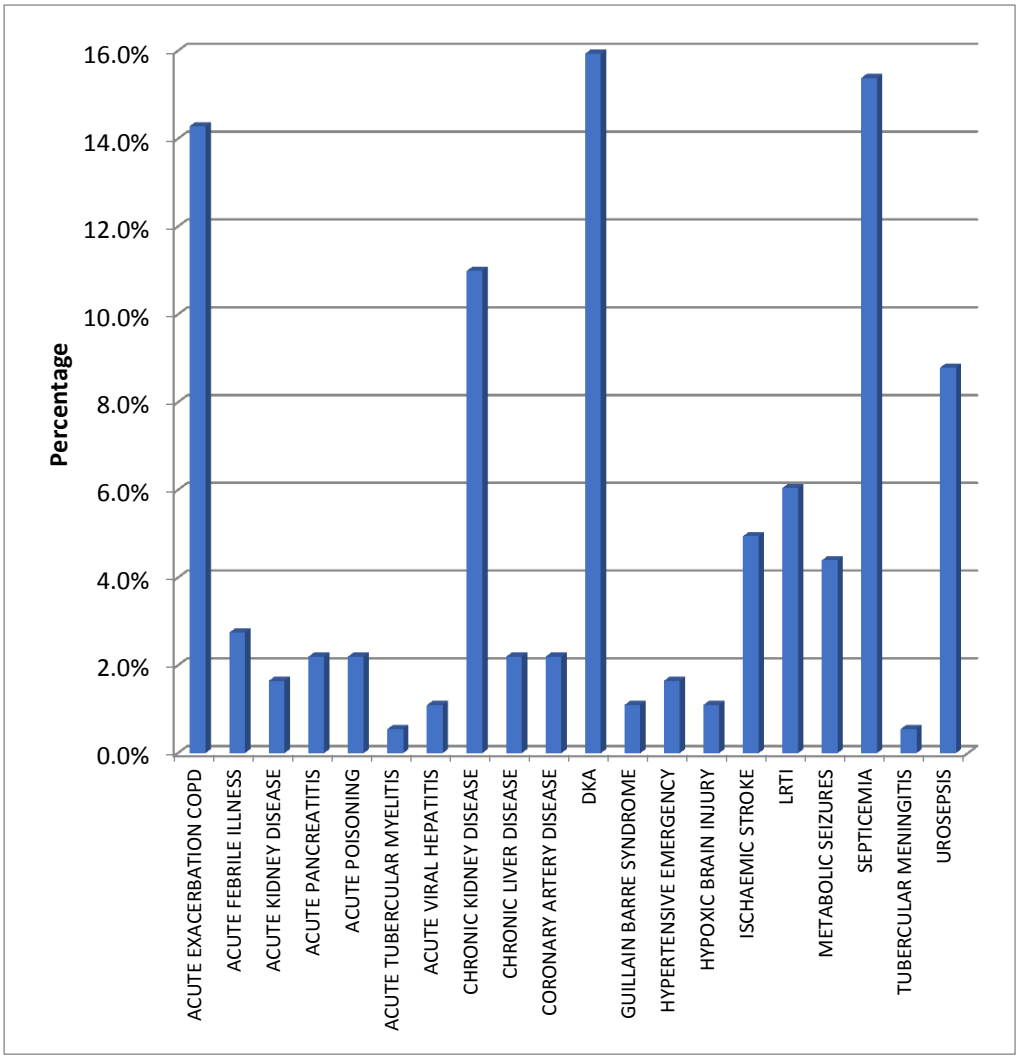

FIGURE 3: DISTRIBUTION OF PATIENTS ACCORDING TO DIAGNOSIS

TABLE 4: DISTRIBUTION OF PATIENTS ACCORDING TO COMORBIDITIES

Comorbidity	No. of cases	Percentage
CKD	22	12.1%
DM	88	48.4%
COPD	32	17.6%
HTN	71	39.0%
CAD	6	3.3%
COPD	32	17.6%
CLD	3	1.6%
ASCITES	2	1.1%
HCV	1	0.5%
HIV	1	0.5%
HYPOTHYROIDISM	1	0.5%
PHTN	1	0.5%

- Diabetes Mellitus (DM) was the most common comorbidity, seen in 88 patients (48.4%) followed by Hypertension (HTN) in 71 patients (39%).
- Chronic Obstructive Pulmonary Disease (COPD) was found in 32 cases, which made up 17.6% of the group.
- Chronic Kidney Disease (CKD) affected 22 patients, comprising 12.1% of the patients.
- Other less common comorbidities included Coronary Artery Disease (CAD), which was seen in 6 patients (3.3%), and Chronic Liver Disease (CLD), which occurred in 3 patients (1.6%).
- Ascites was reported in 2 patients (1.1%), making up 1.1%, while Hepatitis C Virus (HCV), Human Immunodeficiency Virus (HIV), Hypothyroidism, and Pulmonary Hypertension (PHTN) were noted in 1 patient each, contributing 0.5% respectively to the total.

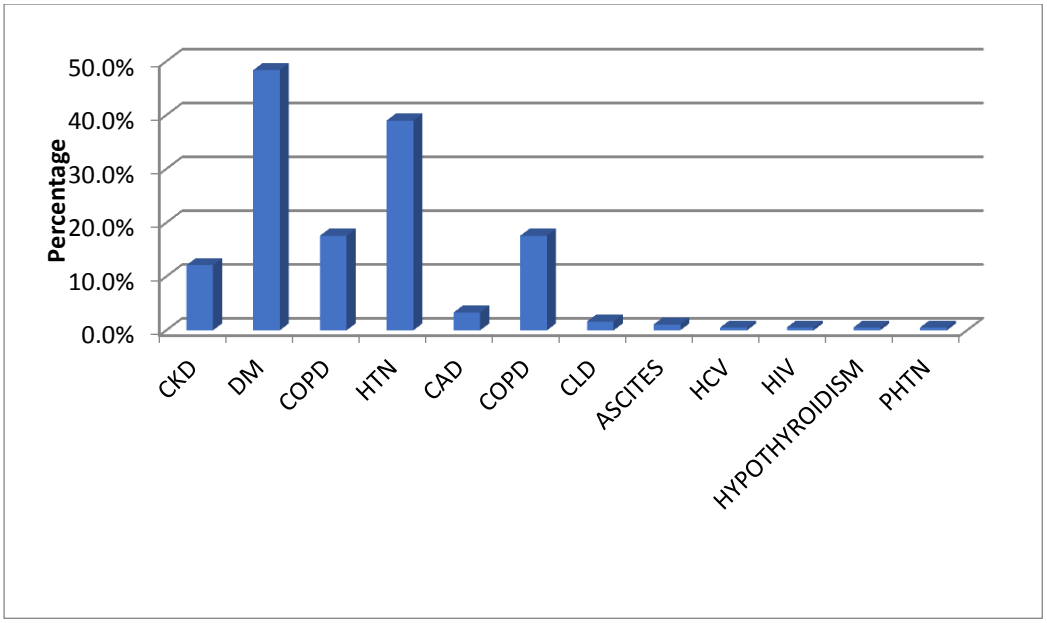

FIGURE 4: DISTRIBUTION OF PATIENTS ACCORDING TO COMORBIDITIES

TABLE 5: DISTRIBUTION OF PRESSURE SORE PATIENTS BASED UPON THE BRADEN SCALE

Braden scale group	No. of cases	Percentage
< 9(very high risk)	0	0.0%
10-12(high risk)	1	0.5%
13-14(moderate risk)	35	19.2%
15-18(mild risk)	146	80.2%
Total	182	100.0%

- Out of the total 182 patients, 1 patient (0.5%) was in the Braden Scale Group 10-12, 35 patients (19.2%) were in the Braden Scale Group 13-14, and 146 patients (80.2%) were in the Braden Scale Group > 15.
- There was no patient in <9 Braden scale group.

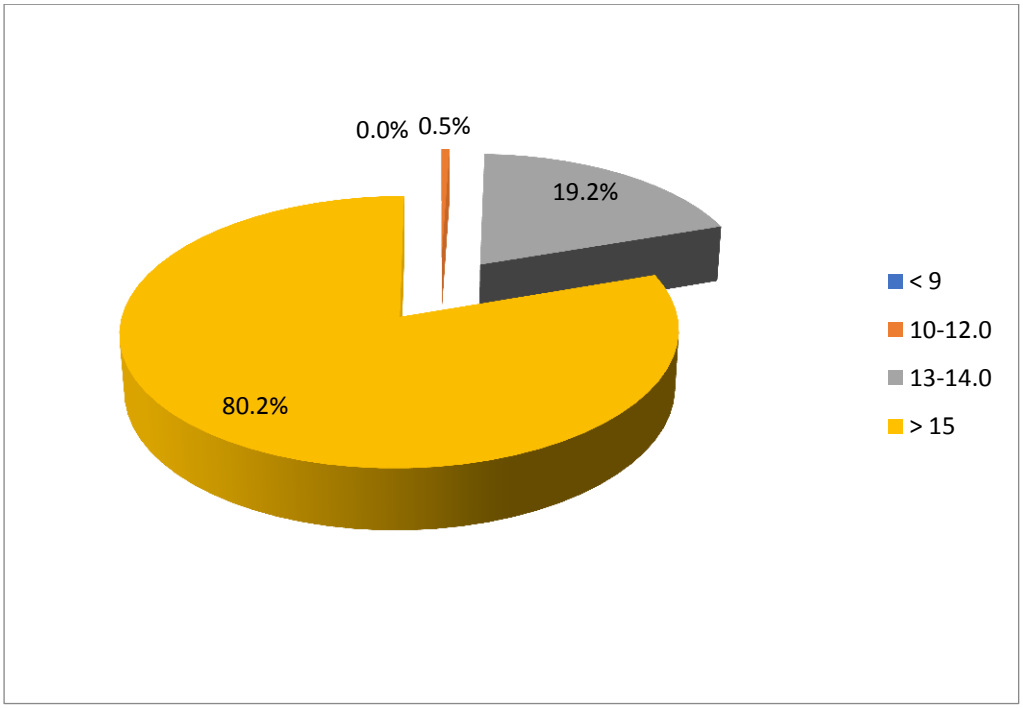

FIGURE 5: DISTRIBUTION OF PRESSURE SORE PATIENTS BASED UPON THE BRADEN SCALE

TABLE 6: DISTRIBUTION OF PATIENTS BASED ON HEMOGLOBIN (HB) LEVELS

Hb(gm/dl)	No. of cases	Percentage
< 8	43	23.6%
> 8	139	76.4%
Total	182	100.0%

- Out of the 182 patients, 43 patients (23.6%) had hemoglobin (Hb) level below 8gm/dl, while 139 patients (76.4%) had hemoglobin level above 8gm/dl.

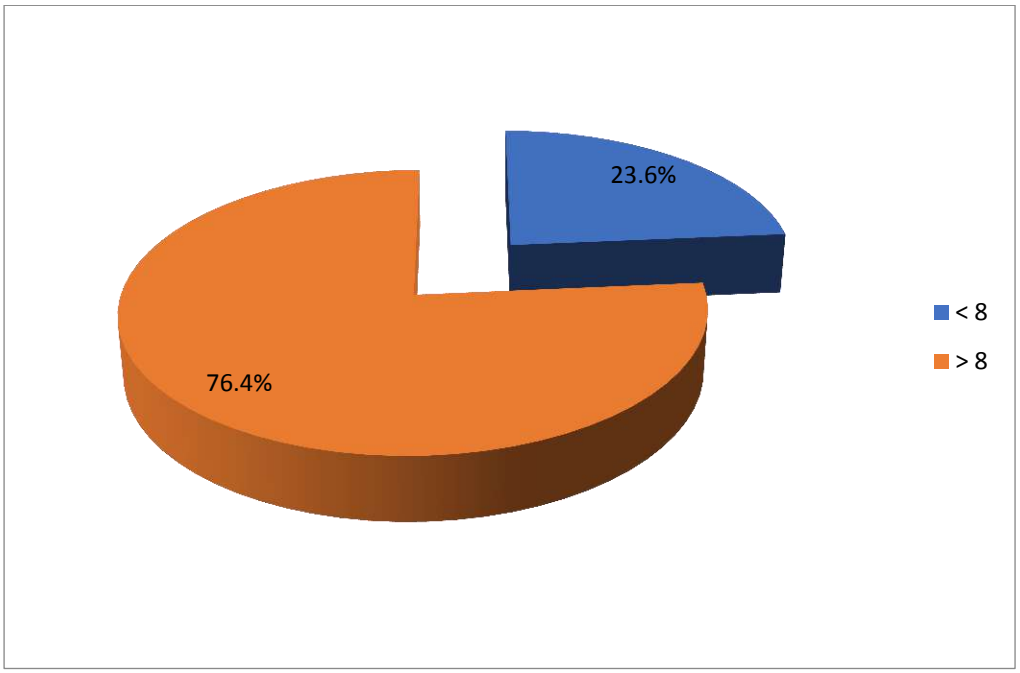

FIGURE 6: DISTRIBUTION OF PATIENTS BASED ON HEMOGLOBIN (HB) LEVELS

TABLE 7: DISTRIBUTION OF PATIENTS BASED ON PLATELET COUNT

Platelets(10^9/l)	No. of cases	Percentage
< 150	113	62.1%
> 150	69	37.9%
Total	182	100.0%

- Out of total 182 patients, 113 patients (62.1%) had platelet counts below 150, while 69 patients (37.9%) had platelet counts above 150.

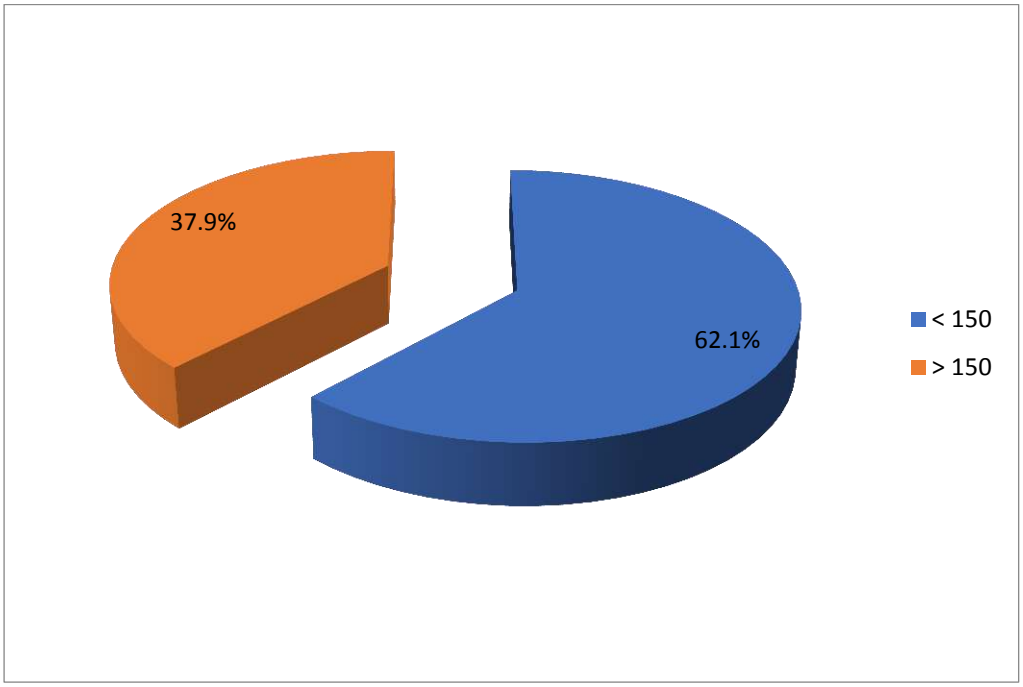

FIGURE 7: DISTRIBUTION OF PATIENTS BASED ON PLATELET COUNT

TABLE 8: DISTRIBUTION OF PATIENTS BASED ON ALBUMIN LEVELS

Albumin(g/dl)	No. of cases	Percentage
< 3.5	174	95.6%
> 3.5	8	4.4%
Total	182	100.0%

- Out of 182 patients, 174 patients (95.6%) had albumin levels below 3.5gm/dl, while only 8 patients (4.4%) had albumin levels above 3.5gm/dl.

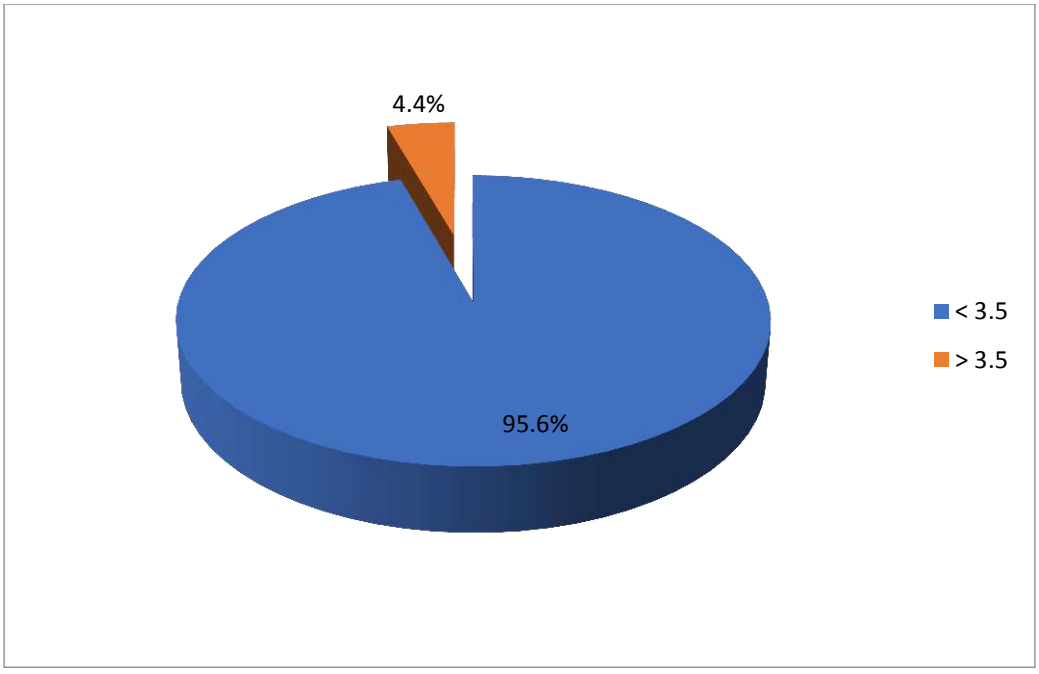

FIGURE 8: DISTRIBUTION OF PATIENTS BASED ON ALBUMIN LEVELS

TABLE 9: DISTRIBUTION OF PATIENTS BASED ON DIRECT BILIRUBIN LEVELS

Direct bilirubin(mg/dl)	No. of cases	Percentage
< 0.2	1	0.5%
> 0.2	181	99.5%
Total	182	100.0%

- Out of 182 cases, 181 patients (99.5%) had direct bilirubin levels above 0.2 mg/dL, while only 1 patient (0.5%) had direct bilirubin levels below 0.2 mg/dL.
- The majority of the cases (99.5%) had direct bilirubin levels more than 0.2 mg/dL.

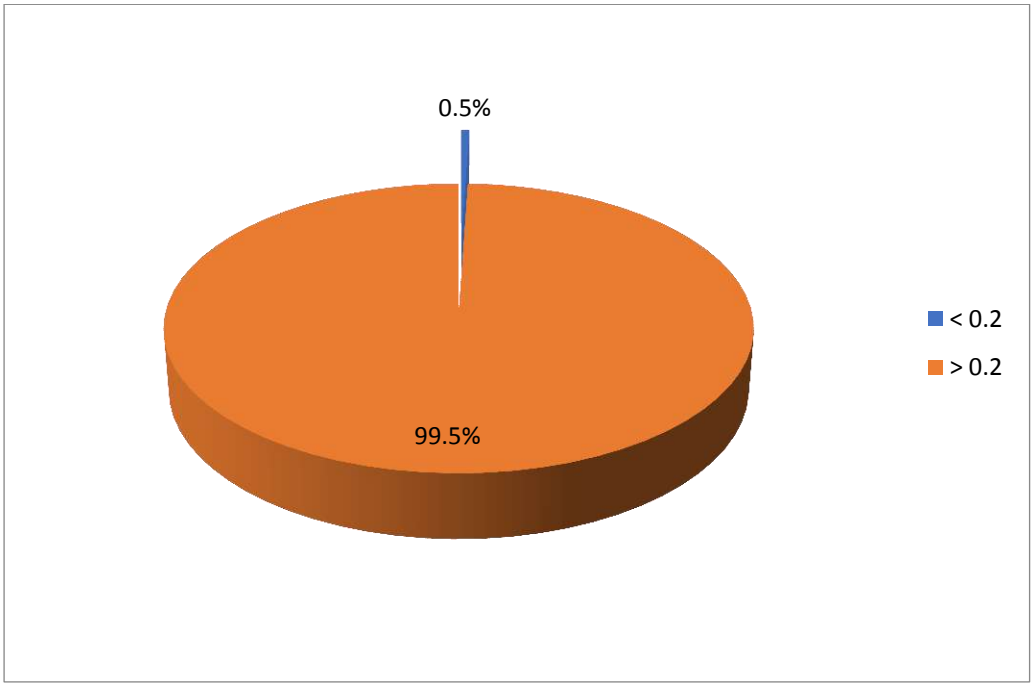

FIGURE 9: DISTRIBUTION OF PATIENTS BASED ON DIRECT BILIRUBIN LEVELS

TABLE 10: DISTRIBUTION OF PATIENTS BASED ON INDIRECT BILIRUBIN LEVELS

Indirect bilirubin (mg/dl)	No. of cases	Percentage
< 0.8	126	69.2%
> 0.8	56	30.8%
Total	182	100.0%

- Out of 182 patients, 56 patients (30.8%) had indirect bilirubin >0.8mg/dL.
- While 126 patients (69.2%) of the total cases had indirect bilirubin <0.8mg/dL.

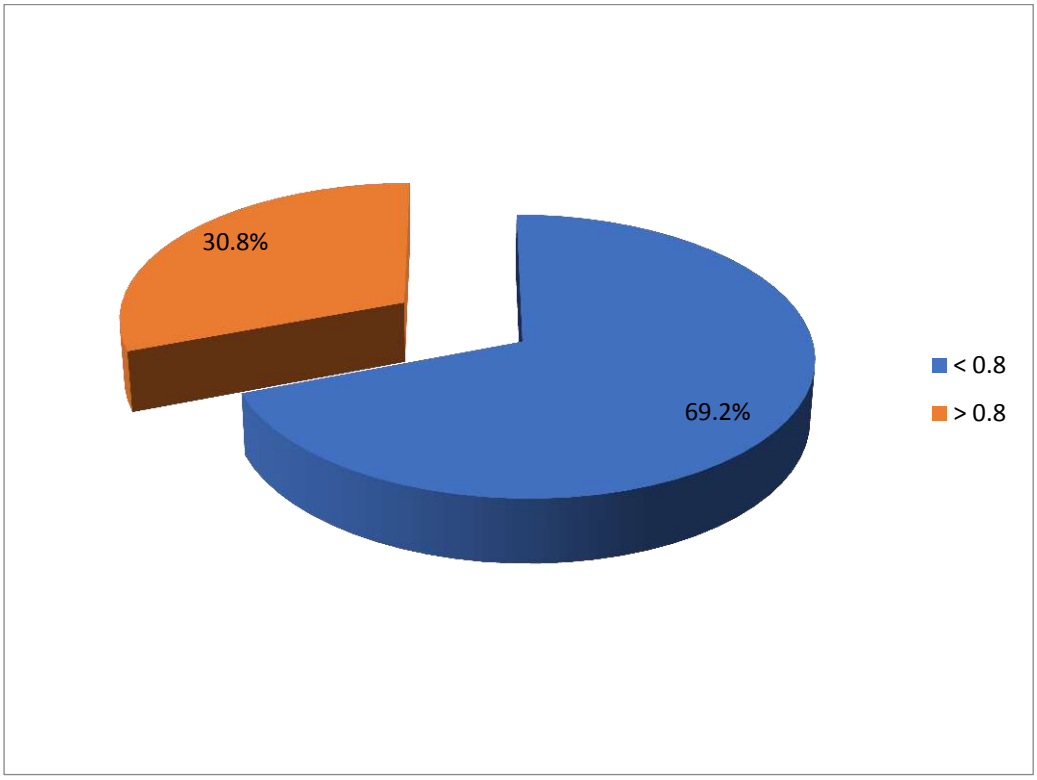

FIGURE 10: DISTRIBUTION OF PATIENTS BASED ON INDIRECT BILIRUBIN LEVELS

TABLE 11: DISTRIBUTION OF PATIENTS BASED ON CREATININE LEVELS

Creatinine (mg/dl)	No. of cases	Percentage
< 1.2	33	18.1%
> 1.2	149	81.9%
Total	182	100.0%

- The majority of the patients i.e.149 patients (81.9%) had creatinine levels greater than 1.2 mg/dL.
- Only 33 patients (18.1%) had creatinine levels less than 1.2 mg/dL, which lies within the normal range for most individuals.

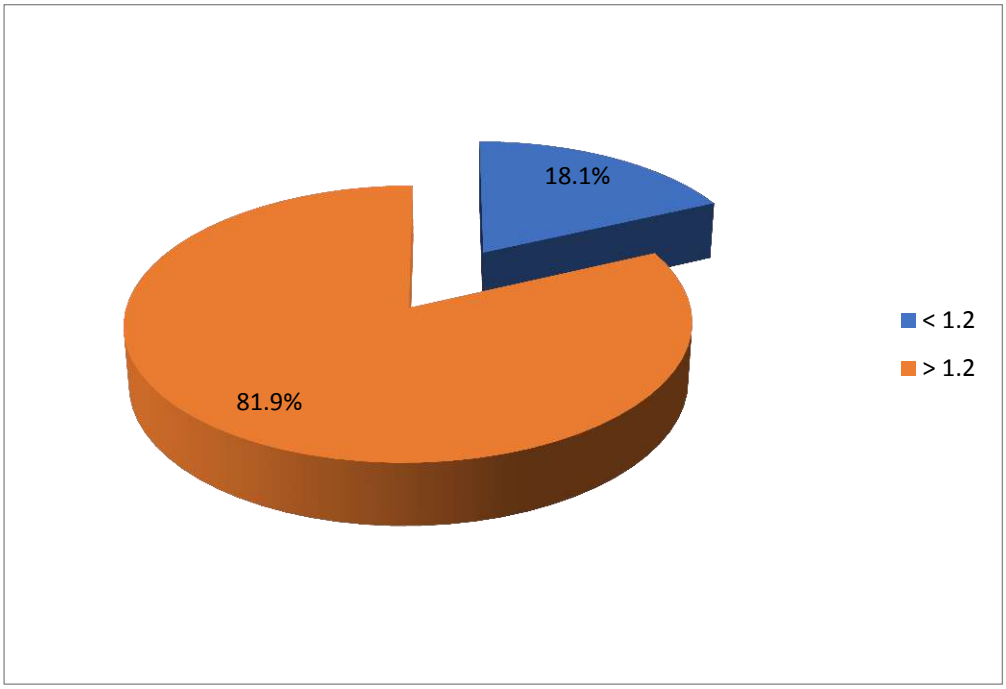

FIGURE 11: DISTRIBUTION OF PATIENTS BASED ON CREATININE LEVELS

TABLE 12: DISTRIBUTION OF PATIENTS BASED ON THE PRESENCE OR ABSENCE OF PRESSURE SORES

Pressure sore	No. of cases	Percentage
NO	110	60.4%
YES	72	39.6%
Total	182	100.0%

- Out of the total 182 patients, 110 patients (60.4%) did not develop pressure sores, while 72 patients (39.6%) developed pressure sores.

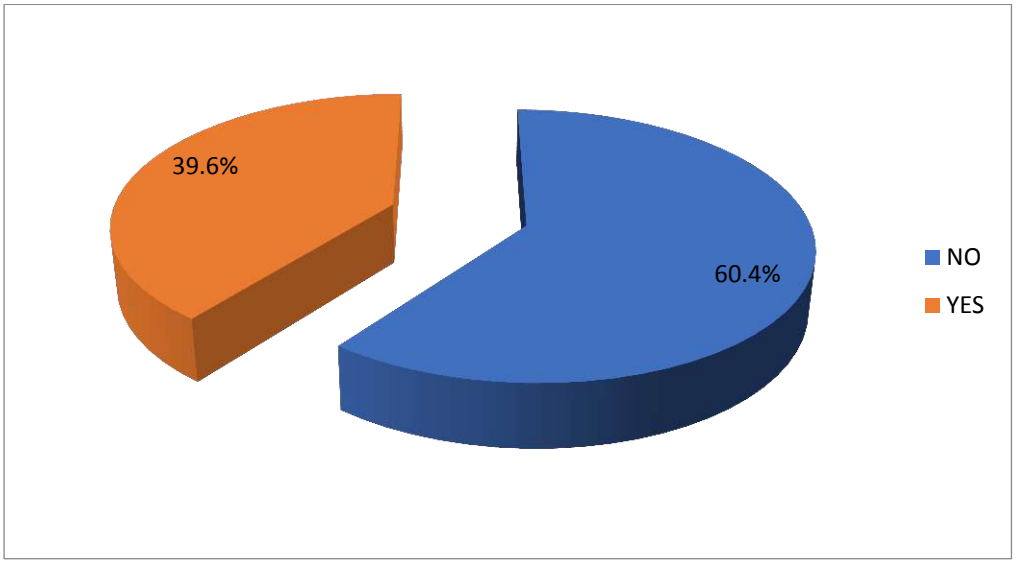

FIGURE 12: DISTRIBUTION OF PATIENTS BASED ON THE PRESENCE OR ABSENCE OF PRESSURE SORES

TABLE 13: DISTRIBUTION OF PATIENTS BASED ON THE STAGES OF PRESSURE SORES

Stage of Pressure sore	No. of cases	Percentage
1	8	11.1%
2	57	79.2%
3	4	5.6%
Unstageable	3	4.2%
Total	72	100.0%

- Among the 72 patients with pressure sores, 8 patients (11.1%) were classified as Stage 1, 57 patients (79.2%) as Stage 2, 4 patients (5.6%) as Stage 3, and 3 patients (4.2%) as unstageable.

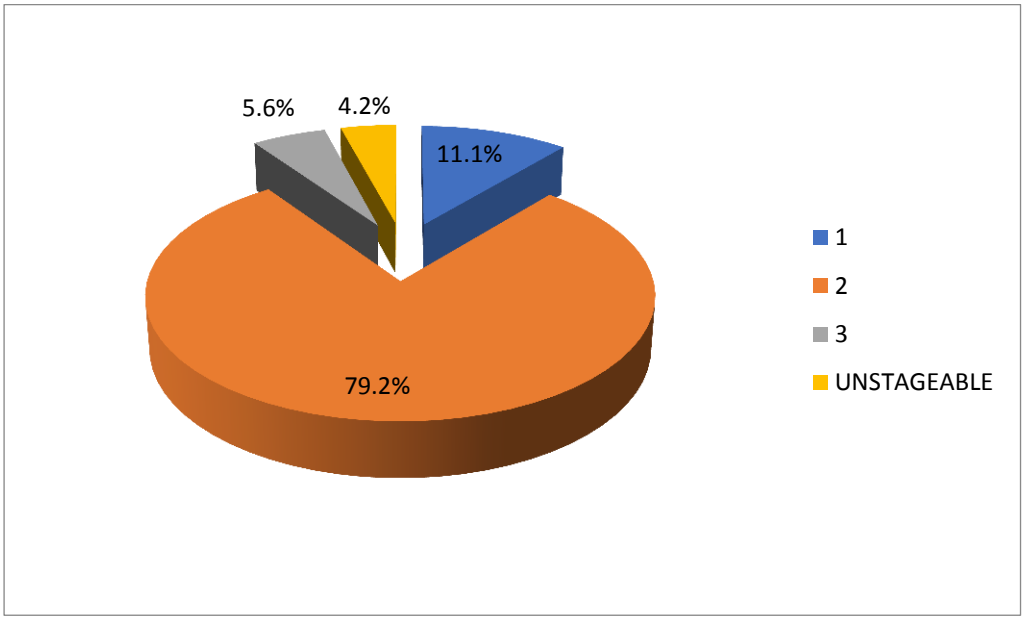

FIGURE 13: DISTRIBUTION OF PATIENTS BASED ON THE STAGES OF PRESSURE SORES

TABLE 14: DISTRIBUTION OF PRESSURE SORES BASED ON THEIR LOCATION

Location	No. of cases	Percentage
Sacral	70	97.2%
Sacral, occiput	1	1.4%
Sacral, b/l heel	1	1.4%
Total	72	100.0%

- All 72 patients (100%) had pressure sores in the sacral region. 1 patient (1.4%) had additional pressure sore in occipital region and 1 patient (1.4%) had additional pressure sore over both the heels.

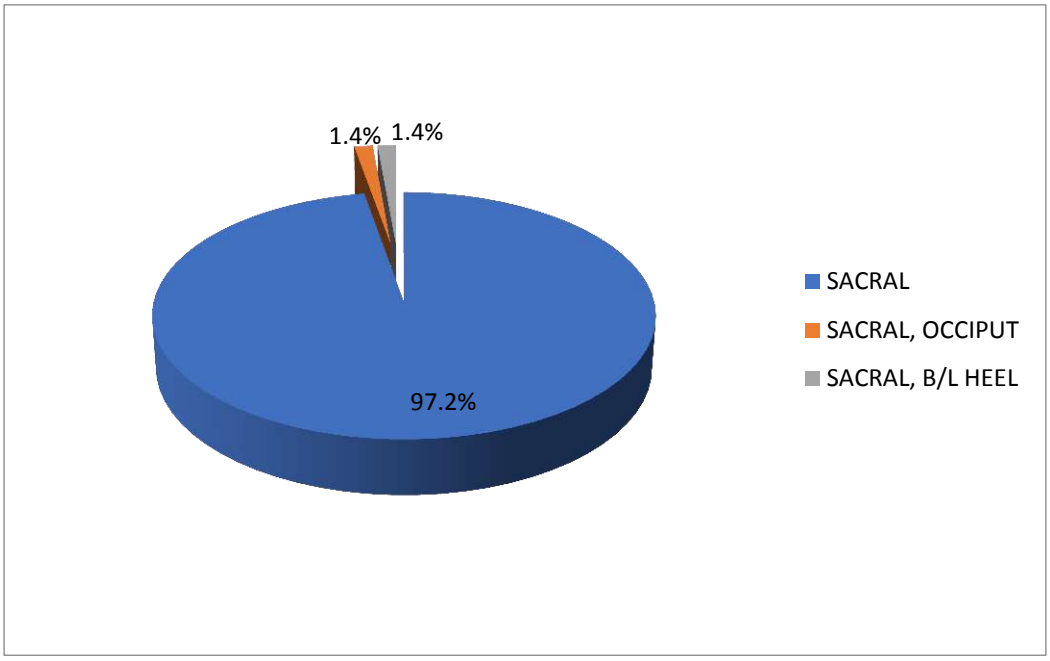

FIGURE 14: DISTRIBUTION OF PRESSURE SORES BASED ON THEIR LOCATION

TABLE 15: DISTRIBUTION OF PRESSURE SORES ACROSS DIFFERENT AGE GROUPS

		Pressure sore				Total	Chi-square value	p-value
		NO		YES				
Age group	< 40	9	8.2%	3	4.2%	12	5.329	0.377
	41-50	18	16.4%	9	12.5%	27		
	51-60	21	19.1%	18	25.0%	39		
	61-70	36	32.7%	18	25.0%	54		
	71-80	22	20.0%	18	25.0%	40		
	> 80	4	3.6%	6	8.3%	10		
Total		110	100.0%	72	100.0%	182		

- In the "< 40" age group, only 3 patients(4.2%) had pressure sores while 9 people (8.2%) did not have pressure sores.
- In the "41-50" age group, only 9 people (12.5%) had pressure sores while 18 people (16.4%) did not have pressure sores.
- In the "51-60" age group, 18 people (25.0%) developed pressure sores while 21 people (19.1%) did not have pressure sores.
- In the "61-70" age group, 18 people (25.0%) had pressure sores while 36 people (32.7%) did not have pressure sores.
- In the "71-80" age group, 18 people (25.0%) had pressure sores and on the other hand, 22 people (20.0%) did not have pressure sores.
- In the "> 80" age group, 6 people (8.3%) had pressure sores while 4 people (3.6%) did not have pressure sores.
- The statistical analysis revealed a chi-square value of 5.329 and a p-value of 0.377.
- Since the p-value exceeds 0.05, this indicates that there is no statistically significant association between age group and the pressure sores occurence.

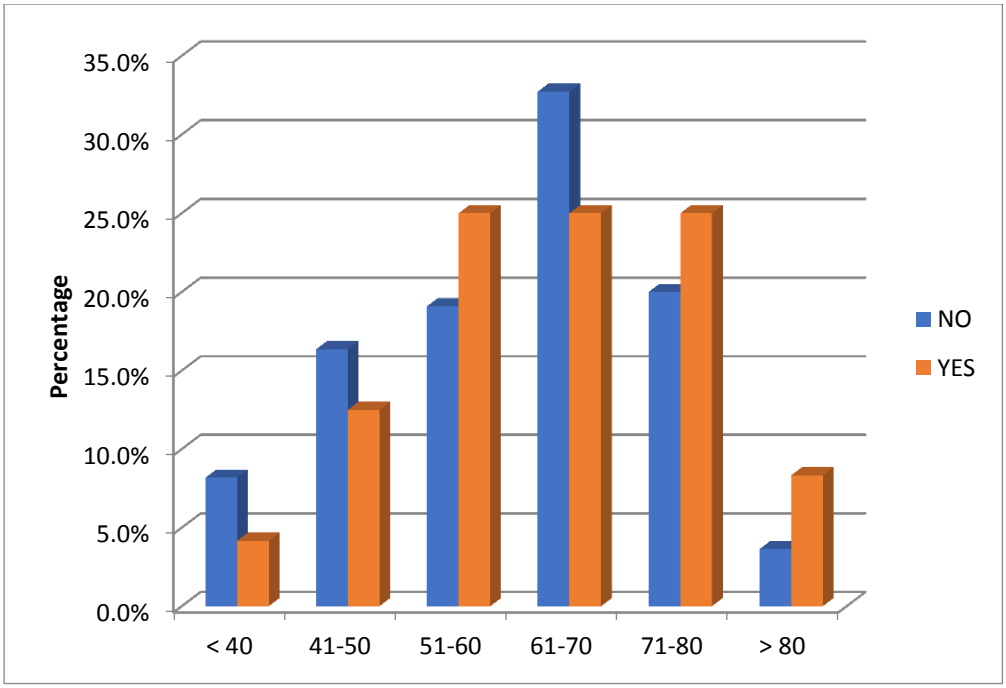

FIGURE 15: DISTRIBUTION OF PRESSURE SORES ACROSS DIFFERENT AGE GROUPS

TABLE 16: DISTRIBUTION OF PRESSURE SORES BY SEX

		Pressure sore				Total	Chi-square value	p-value
		NO		YES				
Sex	F	40	36.4%	31	43.1%	71	0.819	0.437
	M	70	63.6%	41	56.9%	111		
Total		110	100.0%	72	100.0%	182		

- Among 71 female patients, 31 patients (43.1%) developed pressure sores, while 40 patients (36.4%) did not.
- Among 111 male patients, 41 patients (56.9%) developed pressure sores, while 70 patients (63.6%) did not.
- The proportion of patients with pressure sores is slightly higher among males i.e. 56.9% than among the female patients i.e.43.1%.
- The statistical analysis yielded a chi-square value of 0.819 and a p-value of 0.437.
- Since the p-value is greater than 0.05, it suggests that there is no statistically significant association between sex and pressure sores occurence.

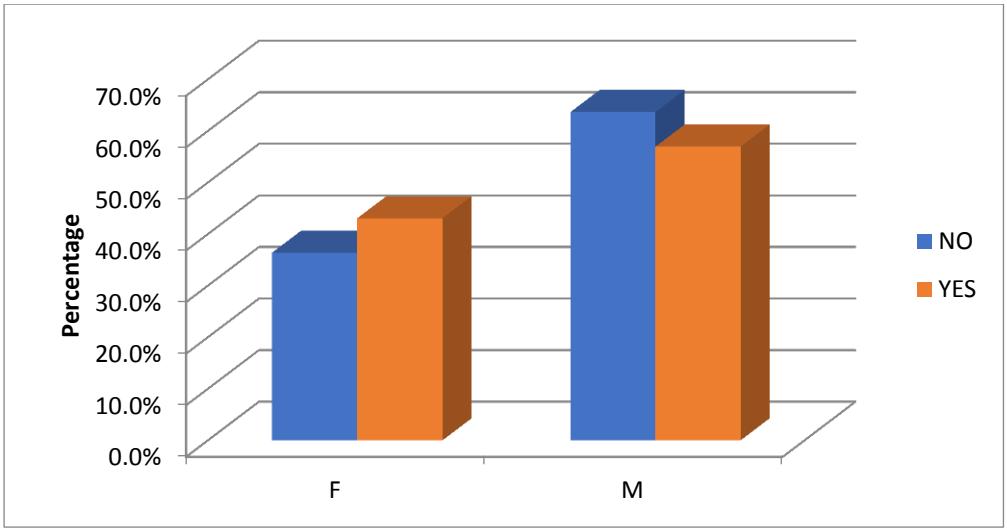

FIGURE 16: DISTRIBUTION OF PRESSURE SORES BY SEX

TABLE 17: RELATIONSHIP BETWEEN BRADEN SCALE AND THE PRESENCE OF PRESSURE SORES

		Pressure sore				Total	Chi-square value	p-value
		NO		YES				
Braden scale group	10-12	0	0.0%	1	1.4%	1	9.367	0.009
	13-14	14	12.7%	21	29.2%	35		
	> 15	96	87.3%	50	69.4%	146		
Total		110	100.0%	72	100.0%	182		

- In the Braden Scale group categorized as "10-12" (high risk), only 1 patient (1.4%) developed pressure sore.
- In the "13-14" group (moderate risk), 21 patients (29.2%) developed pressure sore and 14 patients (12.7%) did not develop pressure sores, with a total of 35 patients in this group.
- In the "> 15" group (low risk), 50 patients (69.4%) developed pressure sore and 96 patients (87.3%) did not develop pressure sore, with a total of 146 patients in this group.
- Statistical analysis revealed a chi-square value of 9.367 and a p-value of 0.009.
- The p-value of 0.009 is less than 0.05, indicating that there is a statistically significant association between Braden Scale and pressure sores occurrence.

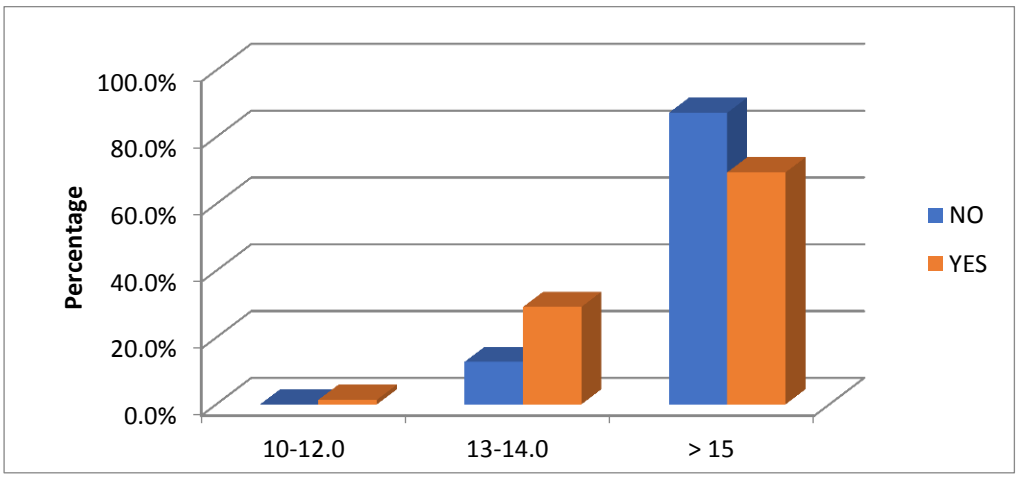

FIGURE 17: RELATIONSHIP BETWEEN BRADEN SCALE AND THE PRESENCE OF PRESSURE SORES

TABLE 18: RELATIONSHIP BETWEEN HEMOGLOBIN (HB) LEVELS AND THE PRESENCE OF PRESSURE SORES

		Pressure sore				Total	Chi-square value	p-value
		NO		YES				
Hb(gm/dl)	< 8	16	14.5%	27	37.5%	43	12.707	0.001
	> 8	94	85.5%	45	62.5%	139		
Total		110	100%	72	100%	182		

- The groups were divided into those with Hb levels "< 8gm/dl" and "> 8gm/dl".
- In the group with hemoglobin (Hb) levels less than 8gm/dl, 27 patients (37.5%) developed pressure sores and 16 patients (14.5%) did not develop pressure sores, for a total of 43 patients in this group.
- In the group with Hb levels greater than 8gm/dl, 45 patients (62.5%) developed pressure sores and 94 patients (85.5%) did not develop pressure sores, for a total of 139 patients in this group.
- Statistical analysis yielded a chi-square value of 12.707 and a p-value of 0.001.
- Since p-value is <0.05, it indicates a statistically significant association between Hb levels and pressure sores occurrence.

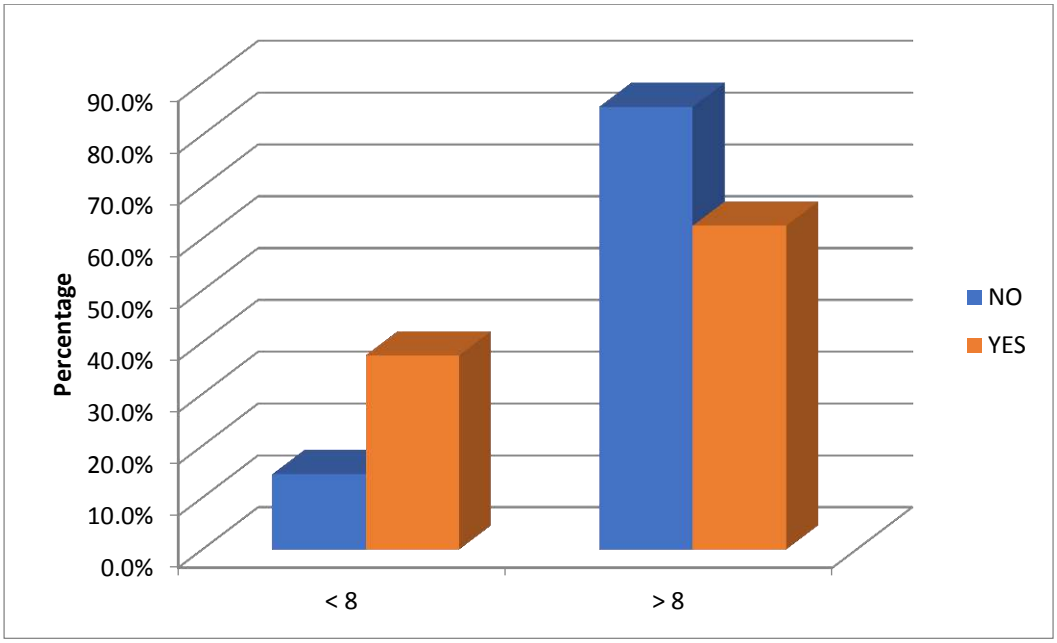

FIGURE 18: RELATIONSHIP BETWEEN HEMOGLOBIN (HB) LEVELS AND THE PRESENCE OF PRESSURE SORES

TABLE 19: RELATIONSHIP BETWEEN PLATELET LEVELS AND THE PRESENCE OF PRESSURE SORES

		Pressure sore				Total	Chi-square value	p-value
		NO		YES				
Platelets ($x10^{9/l}$)	< 150	59	53.6%	54	75.0%	113	8.438	0.005
	> 150	51	46.4%	18	25.0%	69		
Total		110	100%	72	100%	182		

- In the group with platelet levels below 150($x10^{9/l}$), 54 patients (75.0%) developed pressure sores while 59 patients (53.6%) did not develop pressure sores, with a total of 113 patients in this group.
- In the group with platelet levels above 150($x10^{9/l}$), 18 patients (25.0%) developed while 51 patients (46.4%) did not develop pressure sores, with a total of 69 patients in this group.
- The statistical analysis shows a chi-square value of 8.438 and a p-value of 0.005.
- Since the p-value is <0.05, it indicates a statistically significant association between platelet levels and pressure occurrence.

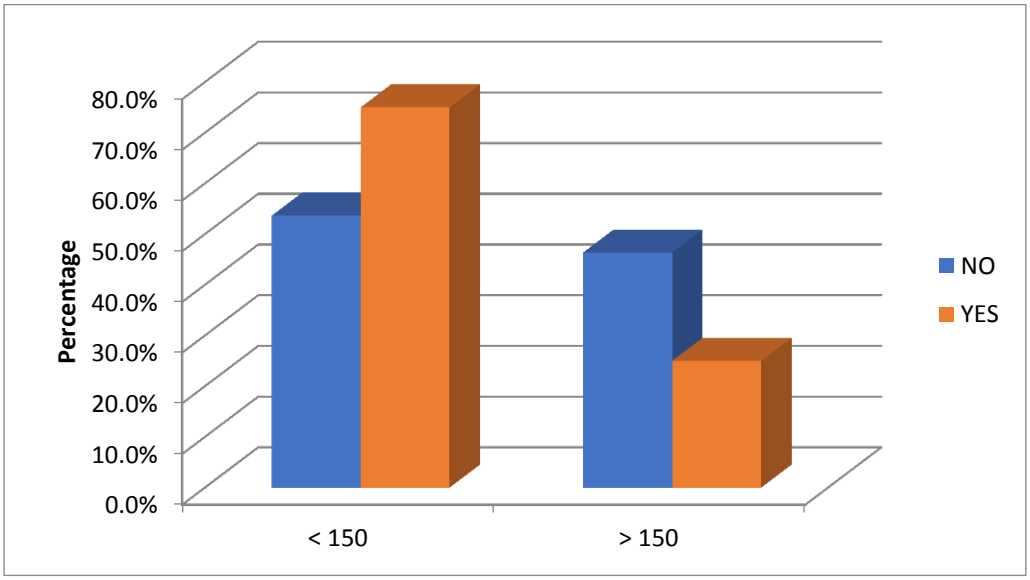

FIGURE 19: RELATIONSHIP BETWEEN PLATELET LEVELS AND THE PRESENCE OF PRESSURE SORES

TABLE 20: RELATIONSHIP BETWEEN ALBUMIN LEVELS AND THE PRESENCE OF PRESSURE SORES

		Pressure sore				Total	Chi-square value	p-value
		NO		YES				
Albumin (mg/dl)	< 3.5	104	94.5%	70	97.2%	174	0.742	0.482
	> 3.5	6	5.5%	2	2.8%	8		
Total		110	100.0%	72	100.0%	182		

- Albumin levels were categorized into two groups: less than 3.5mg/dl and greater than 3.5gm/dl.
- Among those with albumin levels below 3.5mg/dl, 70 patients (97.2%) developed pressure sores while 104 patients (94.5%) did not develop pressure sores, with a total of 174 patients in this group.
- In the group with albumin levels above 3.5mg/dl, only 2 patients (2.8%) developed pressure sores while 6 patients (5.5%) did not develop pressure sores, with a total of 8 patients in this group.
- The chi-square test yielded a value of 0.742 and a p-value of 0.482.
- Since the p-value is >0.05, these results indicate no statistically significant association between albumin levels and the occurrence of pressure sores.

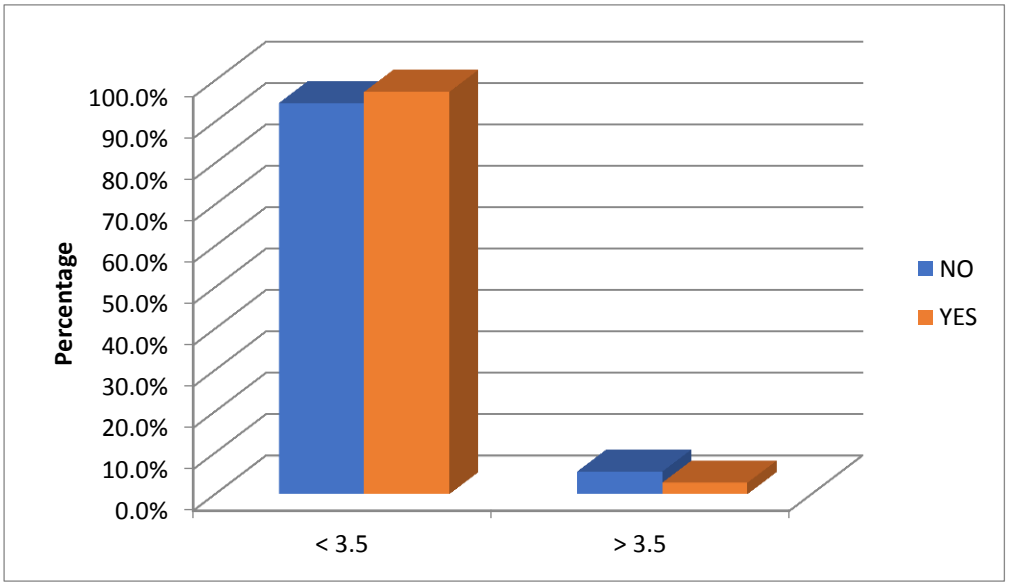

FIGURE 20: RELATIONSHIP BETWEEN ALBUMIN LEVELS AND THE PRESENCE OF PRESSURE SORES

TABLE 21: RELATIONSHIP BETWEEN DIRECT BILIRUBIN LEVELS AND THE OCCURRENCE OF PRESSURE SORES

		Pressure sore				Total	Chi-square value	p-value
		NO		YES				
Direct bilirubin (mg/dl)	< 0.2	1	0.9%	0	0.0%	1	0.658	0.417
	> 0.2	109	99.1%	72	100.0%	181		
Total		110	100.0%	72	100.0%	182		

- Direct bilirubin levels were categorised into two groups: less than 0.2mg/dl and greater than 0.2mg/dl.
- In the group with direct bilirubin levels above 0.2mg/dl, 72 patients (100%) developed pressure sores while 109 patients (99.1%) did not develop pressure sores, with a total of 181 patients in this group.
- There was only patient with direct bilirubin levels <0.2mg/dl and he did not develop pressure sore.
- The chi-square test resulted in a value of 0.658 with a p-value of 0.417.
- Since the p-value is less than 0.05, it indicates a statistically significant association between direct bilirubin levels and the occurrence of pressure sores.

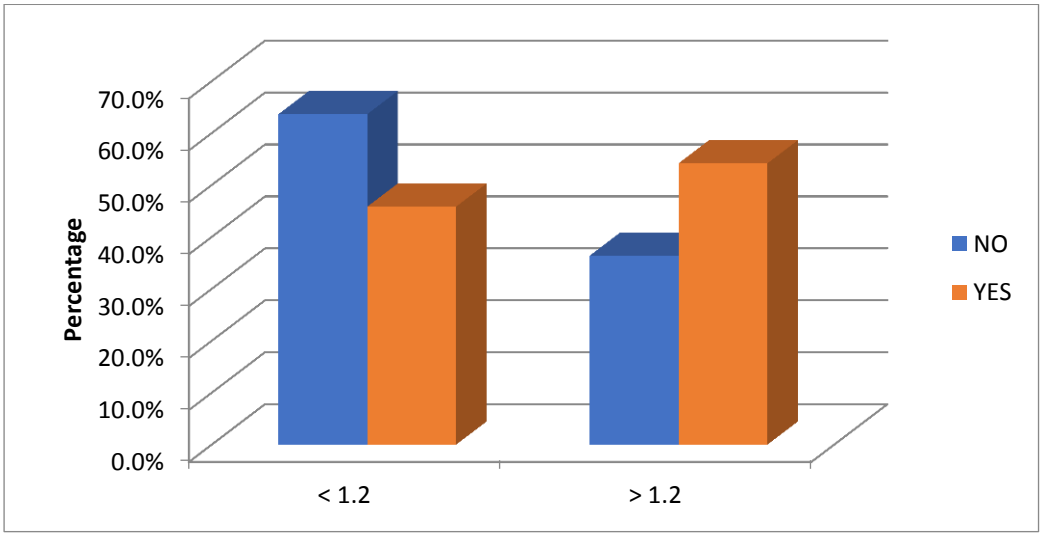

FIGURE 21: RELATIONSHIP BETWEEN DIRECT BILIRUBIN LEVELS AND THE OCCURRENCE OF PRESSURE SORES

TABLE 22: RELATIONSHIP BETWEEN INDIRECT BILIRUBIN LEVELS AND THE OCCURRENCE OF PRESSURE SORES

		Pressure sore				Total	Chi-square value	p-value
		NO		YES				
Indirect bilirubin (mg/dl)	< 0.8	86	78.2%	40	55.6%	126	10.458	0.002
	> 0.8	24	21.8%	32	44.4%	56		
Total		110	100.0%	72	100.0%	182		

- Indirect bilirubin levels were categorized into two groups: less than 0.8mg/dl and greater than 0.8mg/dl.
- In the group with indirect bilirubin levels above 0.8mg/dl, 32 patients (44.4) had pressure sores while 24 patients (21.8%) did not have pressure sores, with a total of 56 patients in this group.
- Among those with indirect bilirubin levels below 0.8mg/dl, 40 patients (55.6%) had pressure sores while 86 patients (78.2%) did not have pressure sores, with a total of 126 patients in this group.
- The chi-square test yielded a value of 10.458 and a p-value of 0.002.
- Sine the p-value is <0.05, it indicates a statistically significant association between indirect bilirubin levels and the occurrence of pressure sores.

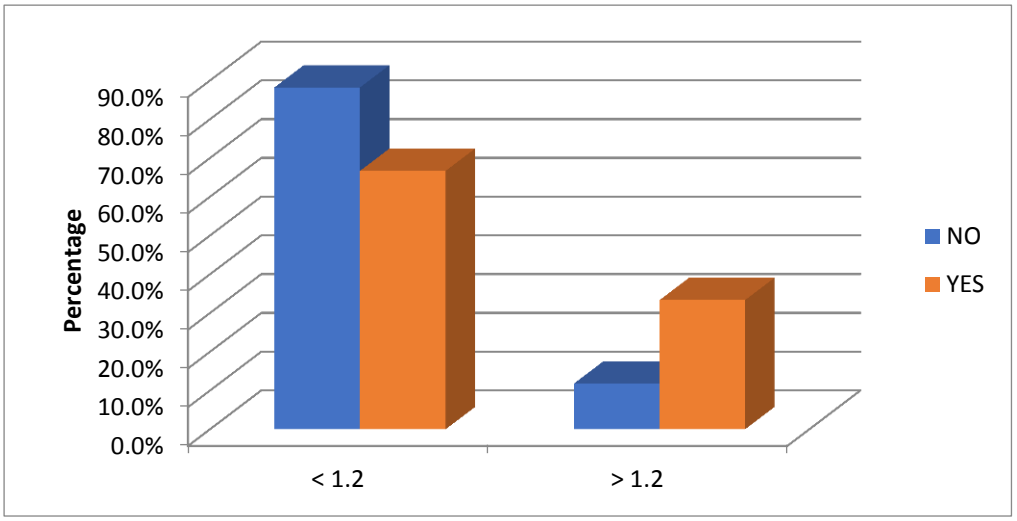

FIGURE 22: RELATIONSHIP BETWEEN INDIRECT BILIRUBIN LEVELS AND THE OCCURRENCE OF PRESSURE SORES

TABLE 23: RELATIONSHIP BETWEEN CREATININE LEVELS AND THE OCCURRENCE OF PRESSURE SORES

		Pressure sore				Total	Chi-square value	p-value
		NO		YES				
Creatinine (mg/dl)	< 1.2	26	23.6%	7	9.7%	33	5.676	0.019
	> 1.2	84	76.4%	65	90.3%	149		
Total		110	100.0%	72	100.0%	182		

- Creatinine levels were categorized into two groups: less than 1.2mg/dl and greater than 1.2mg/dl.
- In the group with creatinine levels above 1.2mg/dl, 65 patients (90.3%) had pressure sores while 84 patients (76.4%) did not have pressure sores, with a total of 149 patients in this group.
- Among individuals with creatinine levels below 1.2mg/dl, 7 patients (9.7%) had pressure sores while 26 patients (23.6%) did not have pressure sores, with a total of 33 patients in this group.
- The chi-square test yielded a value of 5.676 with a p-value of 0.019.
- Since the p-value is <0.05, it indicates a statistically significant association between creatinine levels and the occurrence of pressure sores

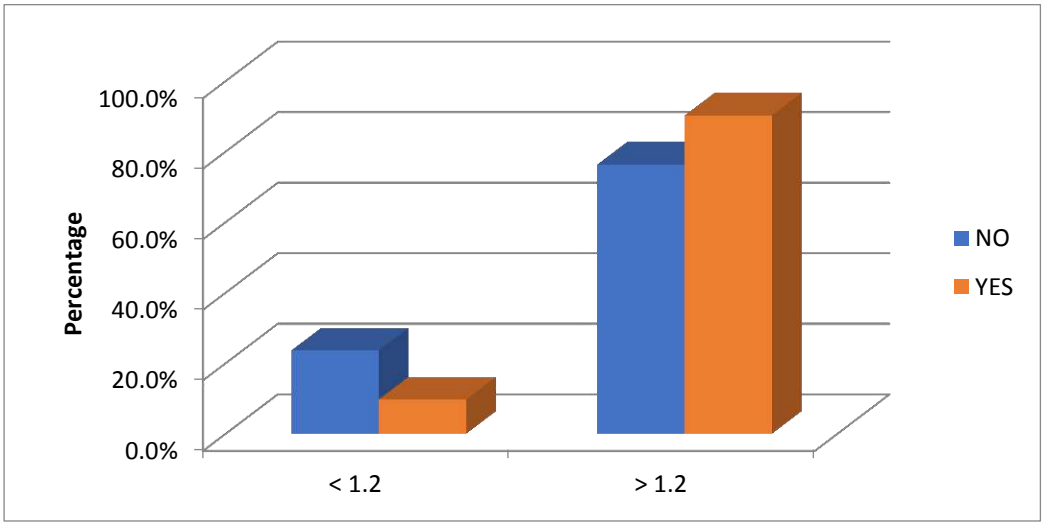

FIGURE 23: RELATIONSHIP BETWEEN CREATININE LEVELS AND THE OCCURRENCE OF PRESSURE SORES

TABLE 24: STATISTICS FOR AGE WITH RESPECT TO THE OCCURRENCE OF PRESSURE SORES

	NO				YES				Z	p-value
	Mean	SD	Median	IQR	Mean	SD	Median	IQR		
Age	61.20	12.91	64	50.75-70	63.67	13.00	66	55.25-74.75	-1.205	0.228

- The mean age for patients with pressure sores was 63.67 years, with a standard deviation of 13.00. The median age was 66 years, with an IQR of 55.25 to 74.75 years.
- For patients without pressure sores showed a mean age of 61.20 years with a standard deviation of 12.91. The median age in this group was 64 years, with an interquartile range (IQR) of 50.75 to 70 years.
- The statistical test results yielded a z-value of -1.205 and a p-value of 0.228.
- Since the p-value is greater than the 0.05, these results indicate that there is no statistically significant difference in age between patients with and without pressure sores.

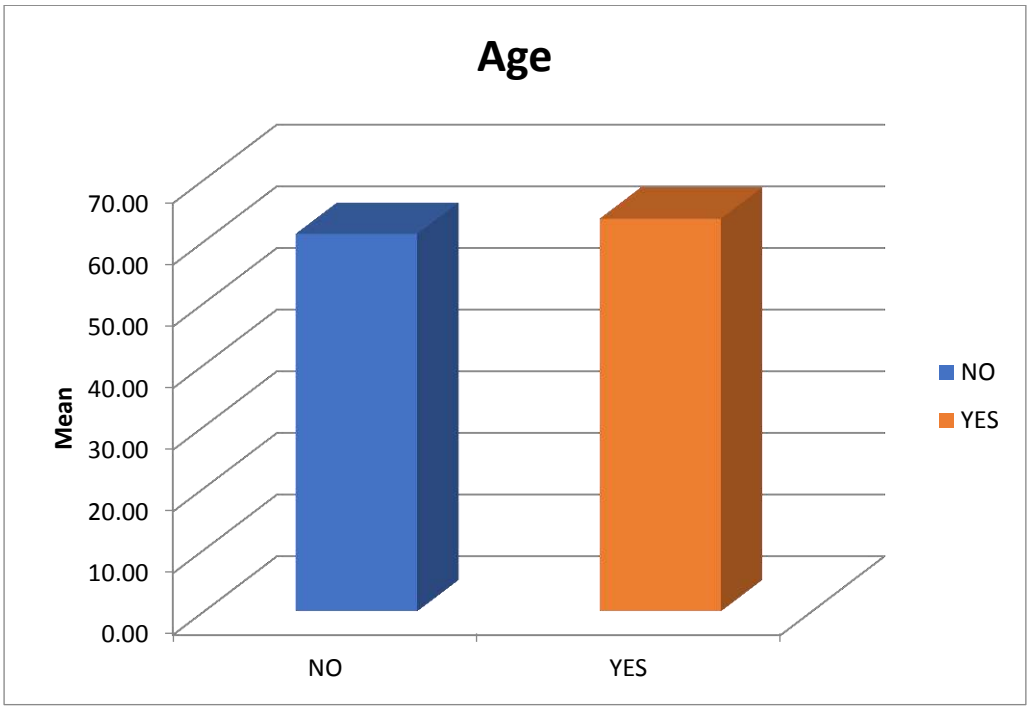

FIGURE 24: STATISTICS FOR AGE WITH RESPECT TO THE OCCURRENCE OF PRESSURE SORES

TABLE 25: STATISTICS FOR THE BRADEN SCALE WITH RESPECT TO THE OCCURRENCE OF PRESSURE SORES

	NO				YES				Z	p-value
	Mean	SD	Median	IQR	Mean	SD	Median	IQR		
Braden scale	16.65	1.38	16	16-18	15.83	1.57	16	14-17	-3.488	0.001

- The mean braden scale score for patients with pressure sores was 15.83 with a standard deviation of 1.57. The median score was 16, with an IQR of 14 to 17.
- For patients without pressure sores, mean braden scale score was 16.65 with a standard deviation of 1.38. The median score for this group was 16, with an interquartile range (IQR) of 16 to 18.
- The statistical test results showed a z-value of -3.488 and a p-value of 0.001.
- Since the p-value is below 0.05, it indicates a statistically significant difference in Braden scale scores between individuals with and without pressure sores.

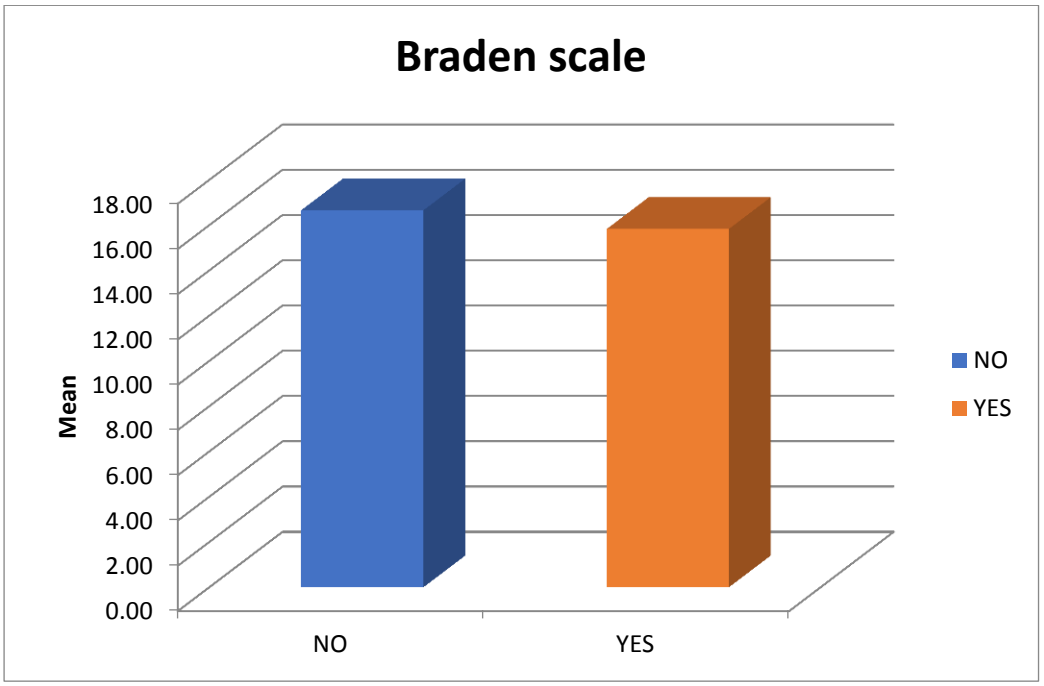

FIGURE 25: STATISTICS FOR THE BRADEN SCALE WITH RESPECT TO THE OCCURRENCE OF PRESSURE SORES

TABLE 26: STATISTICS AND STATISTICAL TEST RESULTS FOR VARIOUS BLOOD MARKERS (HB, PLATELETS, ALBUMIN, DIRECT BILIRUBIN, INDIRECT BILIRUBIN, AND CREATININE) WITH RESPECT TO THE OCCURRENCE OF PRESSURE SORES

	NO				YES				Z	p-value
	Mean	SD	Median	IQR	Mean	SD	Median	IQR		
Hb	12.30	17.74	9.95	8.775-11.025	8.72	1.84	8.6	7.4-9.6	-4.565	0.001
Platelets	153.09	70.12	142	111-178.5	121.04	47.16	111	98-150	-3.623	0.001
Albumin	2.64	0.59	2.75	2.135-3.1	2.18	0.60	2.125	1.7725-2.58	-4.922	0.001
Direct bilirubin	1.67	2.60	0.98	0.79-1.6725	2.87	5.16	1.57	0.9125-2.7075	-2.803	0.005
Indirect bilirubin	0.96	2.12	0.5	0.2175-0.74	1.84	3.83	0.685	0.345-1.66	-3.250	0.001
Creatinine	2.09	1.56	1.7	1.2-2.5175	3.35	1.99	3.1	1.945-3.9	-5.345	0.001

- **Hemoglobin (Hb):** Patients with pressure sores had significantly lower hemoglobin levels (mean: 8.72) as compared to those without pressure sores (mean: 12.30). The p-value of 0.001 indicates a strong association between lower Hb levels and pressure sores.
- **Platelets:** Patients with pressure sores had lower platelet counts (mean: 121.04) compared to those without pressure sores (mean: 153.09). The p-value of 0.001 shows a statistically significant relationship between lower platelet counts and pressure sores occurrence.
- **Albumin:** Those with pressure sores had lower albumin levels (mean: 2.18) compared to those without pressure sores (mean: 2.64). The p-value of 0.001 suggests a strong correlation between low albumin levels and the occurrence of pressure sores.
- **Direct Bilirubin:** Higher direct bilirubin levels were found in patients with pressure sores (mean: 2.87) compared to those without (mean: 1.67), with a p-value of 0.005. This indicates a significant association between higher direct bilirubin levels and pressure sores.
- **Indirect Bilirubin:** Similarly, patients with pressure sores had higher indirect bilirubin levels (mean: 1.84) compared to those without pressure sores (mean: 0.96). The p-value of 0.001 highlights a significant relationship between higher indirect bilirubin levels and pressure sores.
- **Creatinine:** Higher creatinine levels were observed in those with pressure sores (mean: 3.35) compared to those without (mean: 2.09). A p-value of 0.001 suggests a statistically significant association between high creatinine levels and the occurrence of pressure sores.
- All the blood markers showed significant differences between patients with and without pressure sores. Lower levels of hemoglobin, platelets, and albumin, along with higher levels of direct bilirubin, indirect bilirubin, and creatinine, are linked to a greater likelihood of developing pressure sores.

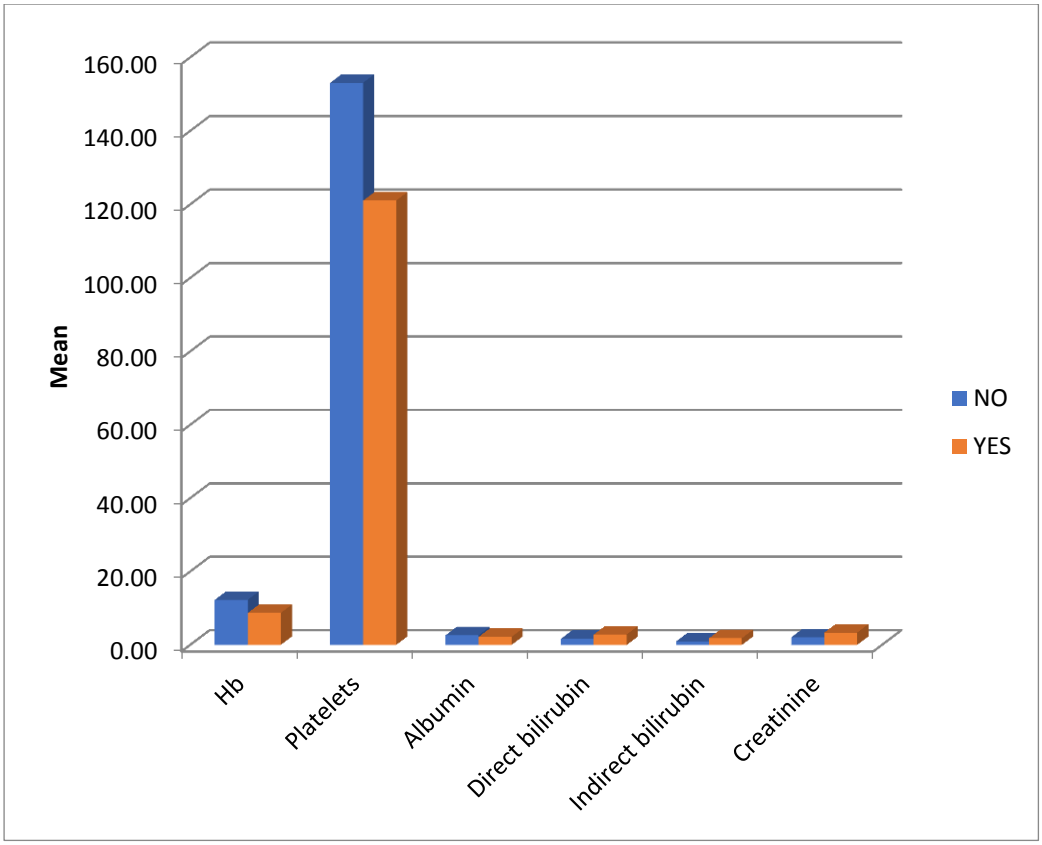

FIGURE 26: STATISTICS AND STATISTICAL TEST RESULTS FOR VARIOUS BLOOD MARKERS (HB, PLATELETS, ALBUMIN, DIRECT BILIRUBIN, INDIRECT BILIRUBIN, AND CREATININE) WITH RESPECT TO THE OCCURRENCE OF PRESSURE SORES

DISCUSSION

Pressure sores develop due to prolonged pressure on skin over bony prominences, reducing blood flow and causing tissue damage. Common sites include the sacrum, ischium, and heels, especially in bedridden or wheel chair bound individuals. Individuals with pressure sores endure significant pain, discomfort, and distress, which extend beyond physical suffering to impact their social, emotional, and mental well-being. It often require prolonged treatment and hospital stays, and are linked to an increased risk of morbidity and mortality. Additionally, pressure sores impose a substantial financial burden on both patients and healthcare system [29].

Assessment tools like the Braden Scale do predict pressure sore occurence, but they may overestimate it, leading to unnecessary interventions. The study highlights the need for improved prediction methods by integrating hematological, and biochemical assessments with existing tools like the Braden Scale to better predict pressure sores in high-risk patients.

Age and Sex: In this study, out of 182 patients, 54 patients (29.7%) are in 61-70 years age group. Males were more frequently affected by pressure sores (61.0%) compared to females (39.0%), indicating a higher prevalence among men.

Our results are in concordance with a 2023 study by Elsorady et al., conducted in Egypt on 191 geriatric patients with pressure sores which reported a mean age of 73.46 ± 8.31 years among those admitted to the hospital [24].

Another study by Miller N et al. on 230 hospitalised patients found that the mean age of those who developed pressure sores was 66.3 years. Of the total patients, 124 (54.4%) were males [30].

A study done by Indraswari et al in Indonesia on 204 patients, concluded that pressure sores developed more commonly among males (56.10%). The age distribution revealed a predominance of patients in the age group of 51 to 60 years (29.27%) [26].

Diagnoses and comorbidities: In the present study, the most common diagnosis among the 182 patients was Diabetic Ketoacidosis (DKA), accounting for 29 patients (15.9%), followed by Septicemia in 28 patients (15.4%) and Acute Exacerbation of COPD in 26 patients (14.3%). The most prevalent comorbidity among the patients was Diabetes Mellitus (DM), affecting 88 patients (48.4%), followed by Hypertension (HTN), present in 71 patients (39%).

According to study done by Elsorady et al, on 191 patients, both DM and CKD were significantly associated with pressure sores. Out of 75 diabetic patients, 26 patients (30.59%) developed pressure sores. P value being <0.05, this was statistically significant [24].

On the contrary, study conducted by Bazalinski et al depicted that most of the patients (56%) were diagnosed with traumatic injuries, and most common comorbidity noted was atherosclerosis i.e. in 58% patients [12].

Incidence of pressure sores: Out of the 182 patients in the present study, 72 patients (39.6%) developed pressure sores while 110 patients (60.4%) did not develop pressure sores. Among those with pressure sores, majority of patients i.e. 57 patients (79.2%) were Stage 2. All 72 cases with pressure sores were located on the sacral area, with 1 patient (1.4%) also developing sores on the occipital region and 1 patient (1.4%) developing sores on both heels.

Study done by Bazalinski et al suggested that incidence of patient developing pressure sores in ICU was 9-11% [12].

A study conducted by Sardo et al revealed incidence of pressure sores to be 5.2-12.3%. And majority of pressure sores were categorised as stage 1 and there were most commonly located at sacral region [31].

A 2023 meta-analysis conducted in Portugal found that stage 1 pressure sores were the most common, followed by stage 2 sores. The majority of the pressure sores were located in the sacral region [31].

Hemoglobin: Present study revealed that there were 43 patients with Hb levels below 8 gm/dl out of which 27 patients (37.5%) developed pressure sores. Patients with pressure sores had lower hemoglobin levels (mean:8.72) as compared to those without pressure sores (mean:12.30). This association was statistically significant, with a chi-

square value of 12.707 and a p-value of 0.001. This indicates a statistically significant association between lower hemoglobin levels and pressure sore occurrence.

However, our results did not align with the study conducted by Elsorady et al on 191 patients, in which only 43 patients (22.5%) developed pressure sores. Mean hemoglobin level was 10.55 ± 2.62 in patients who did not develop pressure sores and 10.04 ± 2.24 in those who did, with specified normal range to be 12-15gm/dl. The p-value of 0.258 indicates that this difference is not statistically significant [24].

Another study done by Bazalinski et al depicted that the mean hemoglobin level in the study group was 10.75 g/dL ± 2.07 g/dL, which is below the normal reference range of 13–17.5 g/dL. Among the participants, 8 individuals (16.0%) had normal hemoglobin levels, while 42 individuals (84.0%) had low levels, with values ranging from 7.10 g/dL to 12.6 g/dL. P-value being 0.428 indicated that low hemoglobin values have no statistically significant association with pressure sores occurrence [12].

Wang et al concluded in a meta analysis done in 2023 that low hemoglobin is a reliable biomarker to predict pressure sore development in patients [23].

Platelet count: In the present study, platelet levels were below 150(x10^9/l) in 113 patients, of whom 54 (75%) developed pressure sores. In contrast, among 69 patients with platelet counts above 150(x10^9/l), only 18 developed pressure sores. The study found a significant correlation between low platelet levels and pressure sore occurrence. The chi-square value for this association was 8.438, with a p-value of 0.005, indicating a statistically significant relationship between lower platelet levels and the occurrence of pressure sores. The mean platelet count in patients with pressure sores was 121.04, compared to 153.09 in those without sores. The p-value of 0.001, being less than 0.05, indicates that low platelet counts are a statistically significant predictor of pressure sore occurence.

Our results were not in concordance with previously conducted studies on role of haematological and biochemical parameters in predicting

pressure sore occurrence. In a study conducted by Elsorady et al., the mean platelet count was 248.29 in patients who developed pressure sores, compared to 253.91 in those who did not. The p-value was 0.797, indicating that the difference was not statistically significant [24].

A study conducted by Dzięgielewska S et al. in 2011, involving 313 patients, revealed that patients with pressure sores had higher mean platelet values compared to those without pressure sores [28].

Albumin: In the present study, majority of the patients were having albumin levels <3.5 mg/dl. Statistical analysis of albumin levels did not show a significant association with pressure sores. Whether albumin levels were below or above 3.5 mg/dl, the occurrence of pressure sores did not differ notably, as reflected in a chi-square value of 0.742 and a p-value of 0.482, indicating no statistical significance. However the mean value of serum albumin in patients with and without pressure sores was 2.18 and 2.64 respectively for which the p-value came out to be <0.05 suggesting that patients who develop pressure sores had relatively lower albumin levels.

In the study conducted by Bazalinski et al in 2021 in 50 patients admitted in ICU setting showed that only 5 patients (10%) of patients had normal albumin levels and concluded that lower concentrations of albumins correspond to a greater risk of pressure sores occurrence [12].

Serra et al. found that lower albumin levels in ICU patients were linked to higher rates of pressure sores [32].

Similarly, Montalcini et al. observed that low albumin levels (<3.1 g/dL) predicted pressure sore development and were associated with higher mortality [27].

Direct bilirubin: In the present study, direct bilirubin levels were divided into two groups: below 0.2 mg/dl and above 0.2 mg/dl. In the group with levels over 0.2 mg/dl, 72 patients (100%) developed pressure sores, while 109 patients (99.1%) did not. Only one patient had direct bilirubin below 0.2 mg/dl and did not develop sores. Although the chi-square value was 0.658, the p-value of 0.417

indicated no statistically significant association between direct bilirubin levels and pressure sores.

Indirect bilirubin: Indirect bilirubin levels were similarly categorised. In patients with levels above 0.8 mg/dl, 32 (44.4%) developed sores, while 24 (21.8%) did not. Among those with levels below 0.8 mg/dl, 40 (55.6%) had sores, and 86 (78.2%) did not. The chi-square value of 10.458 and p-value of 0.002 showed a statistically significant relationship between higher indirect bilirubin levels and pressure sore development.

However, a 2020 study by Cansu et al. in Turkey, conducted in 200 patients, concluded that several factors are associated with an increased risk of developing pressure sores including bilirubin levels [33].

There is limited literature exploring the role of direct and indirect bilirubin levels in predicting pressure sore occurrence.

Creatinine: In the present study, creatinine levels were divided into two groups: below 1.2 mg/dl and above 1.2 mg/dl. Among patients with creatinine levels below 1.2 mg/dl, 7 (9.7%) developed pressure sores, while 26 (23.6%) did not, for a total of 33 patients. In the group with creatinine levels above 1.2 mg/dl, 65 patients (90.3%) had pressure sores, while 84 (76.4%) did not, totalling 149 patients. The chi-square test resulted in a value of 5.676, with a p-value of 0.019, indicating a statistically significant association between higher creatinine levels and the occurrence of pressure sores.

Study done by Elsorady et al stated that mean creatinine value in patients with or without pressure sores is 1.45 and 1.77 respectively. P value being more than 0.05, thus it was not statistical significant [24].

Braden scale: Further in the present study, we compared blood marker values with Braden scale using appropriate statistical methods. In the "10-12" high-risk Braden Scale group, only 1 patient (1.4%) developed a pressure sore. In the "13-14" moderate-risk group, 29.2% of patients developed sores, while 12.7% did not. Among the ">15" low-risk group, 69.4% developed sores, and 87.3% did not. The chi-

square value of 9.367 and a p-value of 0.009 indicate a statistically significant association between Braden Scale scores and the development of pressure sores. The mean Braden Scale score for patients with pressure sores was 15.83, compared to 16.65 for those without sores, showing a statistically significant difference (p-value = 0.001). The z-value of -3.488 further supports this significant difference in Braden Scale scores between the two groups. Patients in higher-risk categories were more likely to develop pressure sores, while those in lower-risk groups had a lower incidence. However, the presence of pressure sores in some low-risk patients suggests that while the Braden Scale is useful, it may not be fully sufficient on its own, indicating the need for additional biomarkers to interpret the development of pressure sores.

A recent study by Elsorady et al. identified a Braden Scale score of ≤14 are at higher risk for developing pressure sores suggesting that patients with scores at or below this threshold are at higher risk for developing pressure sores [24].

There are various studies in literature depicting that Braden assessment scale if considered alone, cannot be a reliable predictor of pressure sore development [34].

The present study revealed that the patients with pressure sores have lower hemoglobin, platelet counts, and albumin levels, along with higher direct bilirubin, indirect bilirubin, and creatinine levels. These factors may help to predict the development of pressures sores in hospitalised patients.

SUMMARY AND CONCLUSION

This study highlights the role of various factors contributing to pressure sore development in hospitalized patients, particularly those at higher risk due to age, comorbidities, and immobility. While tools like the Braden Assessment Scale remain valuable for risk assessment, our findings highlight that they may not be entirely sufficient on their own. The statistically significant associations between pressure sore development and lower hemoglobin, platelet counts, and albumin levels, along with elevated indirect bilirubin and creatinine levels, suggest that integrating these biomarkers could increase the accuracy of pressure sore prediction. The role of haematological and biochemical markers has been examined individually in various studies, but to our knowledge, no study has combined these markers for predicting pressure sores. In the current study, we combined these markers to evaluate their role in predicting the development of pressure sores. However, further research and studies are needed to validate these biomarkers and determine if they offer improved accuracy in predicting pressure sore development.

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Sr. No	MRD No	Diagnosis	Age	Sex	Coomorbidity	Braden scale	Hb	Platelets	Albumin	Direct bilirubin	Indirect bilirubin	Creatinine	Pressure sore	Stage of Pressure sore	Location
1	770752	DKA	80	M	-	16	6.6	48	2.01	2.33	2.17	2.5	NO	NO	-
2	585946	SEPTICEMIA	61	M	DM	13	7.4	154	1.11	1.79	1.63	3.03	YES	2	SACRAL
3	1244084	SEPTICEMIA	69	M	DM,HTN	18	11.8	176	2.01	1.3	0.92	1.01	NO	NO	-
4	1171289	LRTI	60	F	COPD	17	11.1	195	1.35	0.33	0.17	0.58	NO	NO	-
5	1468380	ACUTE PANCREATITIS	75	M	DM	17	7.9	215	2.19	0.51	0.88	1.29	YES	2	SACRAL
6	1464696	ACUTE POISONING	35	M	-	18	12.9	142	2.97	16.09	15.49	11.6	NO	NO	-
7	96264	ACUTE KIDNEY DISEASE	74	M	DM,HTN,CKD	17	7.7	22	1.03	5.45	4.1	3.18	YES	2	SACRAL
8	1468387	SEPTICEMIA	70	M	DM,HTN	16	10.7	163	3.28	0.24	0.08	6.04	NO	NO	-
9	457763	LRTI	59	F	COPD	16	10.3	190	3.34	0.97	0.4	1.39	YES	2	SACRAL
10	1465831	DKA	56	F	DM,CAD	16	9.6	110	2.5	0.99	0.4	1.54	YES	2	SACRAL
11	1461087	ACUTE POISONING	30	M	-	15	10.6	117	3.25	1.11	0.36	0.58	YES	2	SACRAL
12	1458188	ACUTE KIDNEY DISEASE	68	M	-	16	4.6	28	1.56	1.79	1.11	8.41	YES	2	SACRAL
13	1260186	LRTI	75	F	COPD	16	9.8	129	2.55	0.99	0.34	1.23	NO	NO	-
14	1468453	SEPTICEMIA	60	F	HYPOTHYROIDISM	16	8.3	122	2.37	0.9	0.34	1.31	NO	NO	-
15	1235278	HYPOXIC BRAIN INJURY	53	M	-	16	9.6	110	2.46	0.28	0.19	0.68	YES	2	SACRAL
16	1464697	SEPTICEMIA	75	M	DM,HTN,COPD	14	5.4	70	3.26	17.43	8.49	3.91	NO	NO	-
17	224537	ACUTE KIDNEY DISEASE	58	M	CKD	14	9.7	140	1.81	2.19	1.15	7.42	NO	NO	-
18	48758	DKA	78	M	DM,HTN,CAD	15	7.8	80	2.54	0.72	0.55	6.61	NO	NO	-
19	1429287	CHRONIC LIVER DISEASE	65	F	CLD, PHTN	16	8.7	34	1.44	0.96	0.44	1.2	YES	2	SACRAL
20	1429102	ACUTE EXACERBATION COPD	66	M	COPD	17	8.7	187	3.28	0.93	0.6	1.4	NO	NO	-
21	1426898	UROSEPSIS	40	M	-	16	11.6	148	3.04	1.49	0.25	0.55	YES	2	SACRAL
22	1396842	DKA	68	M	DM,HTN	18	6.9	120	2.33	0.91	0.72	3.12	NO	NO	-
23	1249341	ACUTE EXACERBATION COPD	69	F	HTN,COPD	16	7.7	72	2.56	0.91	0.34	2.89	NO	NO	-
24	1425707	SEPTICEMIA	78	F	DM,HTN	18	9	153	1.54	0.68	0.19	2.08	YES	2	SACRAL
25	1393645	ACUTE EXACERBATION COPD	68	F	HTN	14	7.8	86	2.47	0.59	0.53	2.82	YES	2	SACRAL
26	1410671	DKA	55	M	DM	18	11.2	212	3.54	0.3	0.16	0.57	YES	2	SACRAL
27	1428844	UROSEPSIS	66	F	DM	18	9.5	166	1.35	0.93	0.61	11.46	YES	2	SACRAL
28	1426875	ACUTE EXACERBATION COPD	78	M	DM,HTN,COPD	18	13.4	78	1.35	0.55	0.28	5.66	YES	1	SACRAL
29	638369	CHRONIC KIDNEY DISEASE	68	M	CKD	14	7.7	136	3.75	0.39	0.23	2.23	NO	NO	-
30	1429107	UROSEPSIS	82	M	COPD	14	14.9	78	3.54	0.93	0.28	1.5	NO	NO	-
31	1414971	ACUTE EXACERBATION COPD	66	M	COPD	14	8.9	76	1.68	11.92	10.24	1.85	NO	NO	-
32	654942	UROSEPSIS	77	F	-	16	8.9	78	1.66	3.9	3.58	2.48	YES	2	SACRAL
33	771335	ACUTE EXACERBATION COPD	83	M	DM,HTN	14	11	101	2.12	0.96	0.54	4.84	YES	2	SACRAL
34	1426847	ACUTE EXACERBATION COPD	70	F	COPD	16	10.7	205	3.76	1.06	0.72	1.94	NO	NO	-
35	1429441	DKA	66	F	DM,CAD	16	7.6	375	3.12	0.68	0.22	1.9	NO	NO	-
36	37942	CHRONIC KIDNEY DISEASE	81	M	CKD	16	7.4	124	1.53	1.04	0.36	6.12	YES	2	SACRAL
37	1414534	DKA	57	F	DM	16	8.9	252	1.29	0.91	0.76	3.55	YES	2	SACRAL
38	1374586	ACUTE EXACERBATION COPD	78	F	DM,HTN	14	9.2	261	2.17	0.91	0.38	0.62	NO	NO	-
39	1421446	ACUTE EXACERBATION COPD	66	F	DM,HTN	16	9.1	138	3	0.44	0.23	2.52	YES	2	SACRAL
40	560658	DKA	84	M	DM	16	7	151	2.13	2.72	2.29	6.94	YES	2	SACRAL

Sr. No	MRD No	Diagnosis	Age	Sex	Coomorbidity	Braden scale	Hb	Platelets	Albumin	Direct bilirubin	Indirect bilirubin	Creatinine	Pressure sore	Stage of Pressure sore	Location
41	1422831	METABOLIC SEIZURES	82	M	DM,HTN	14	12	158	2.55	0.79	0.12	1.24	NO	NO	-
42	1421876	ACUTE POISONING	31	F	-	16	7.6	102	2.55	17.22	14.86	5.84	YES	1	SACRAL
43	1393645	SEPTICEMIA	68	F	-	16	7.8	86	3.26	0.59	0.53	2.82	NO	NO	-
44	1410672	HYPOXIC BRAIN INJURY	74	M	-	16	8.6	118	2.17	0.94	0.36	3.48	YES	2	SACRAL
45	1422647	CHRONIC KIDNEY DISEASE	52	F	CKD	14	5.3	-	2.16	0.94	0.76	5.19	YES	2	SACRAL
46	414397	CHRONIC LIVER DISEASE	70	M	DM	16	9.4	344	3.12	0.83	0.26	1.64	NO	NO	-
47	615991	CHRONIC LIVER DISEASE	42	M	-	16	6.2	66	1.6	34.19	23.08	3.7	YES	2	SACRAL
48	140826	CHRONIC KIDNEY DISEASE	72	F	CKD	16	10.2	108	2.58	0.98	0.32	2.86	YES	2	SACRAL
49	1414971	ACUTE PANCREATITIS	27	M	-	14	8.9	64	1.68	11.92	10.24	1.85	NO	NO	-
50	654942	UROSEPSIS	58	F	-	16	8.9	78	1.84	3.9	3.58	2.48	YES	2	SACRAL
51	1419241	ACUTE EXACERBATION COPD	71	M	COPD	16	10.7	479	3.16	0.32	0.17	0.62	NO	NO	-
52	907534	CHRONIC KIDNEY DISEASE	67	F	DM,HTN	16	7.6	252	3.08	0.96	0.34	1.33	NO	NO	-
53	1421778	ACUTE FEBRILE ILLNESS	66	F	-	14	9.2	67	2.69	1.39	0.4	0.64	NO	NO	-
54	48758	CHRONIC KIDNEY DISEASE	76	M	CKD	16	8.1	80	2.04	1.17	0.55	6.61	YES	2	SACRAL
55	1302769	DKA	71	M	DM,HTN	14	9.5	150	2.42	0.62	0.44	1.51	YES	2	SACRAL
56	1424478	SEPTICEMIA	43	M	-	14	12.2	220	2.29	24.46	18.9	3.67	YES	2	SACRAL
57	48098	ISCHAEMIC STROKE	67	M	-	14	9.9	100	1.83	0.69	0.36	0.37	NO	NO	-
58	1422831	DKA	73	M	DM	16	12	158	2.74	0.79	0.18	1.24	NO	NO	-
59	1423823	ISCHAEMIC STROKE	62	M	-	14	7.6	82	1.88	1.57	0.78	1.67	NO	NO	-
60	1425218	SEPTICEMIA	50	M	-	16	12.8	133	3.13	0.67	0.09	0.8	NO	NO	-
61	654942	UROSEPSIS	77	F	-	14	8.9	78	1.84	3.9	3.58	2.48	YES	2	SACRAL
62	1425316	HYPERTENSIVE EMERGENCY	65	F	HTN	16	10.8	-	3.22	0.98	0.34	1.42	NO	NO	-
63	907884	CHRONIC KIDNEY DISEASE	67	F	CKD	16	7.6	252	2.58	0.98	0.71	1.33	NO	NO	-
64	1424503	ACUTE EXACERBATION COPD	74	F	-	16	10	239	3.41	0.91	0.72	1.47	NO	NO	-
65	1350955	HYPERTENSIVE EMERGENCY	34	M	HTN	17	7.5	62	3.65	0.95	0.39	3.04	NO	NO	-
66	1425846	METABOLIC SEIZURES	38	F	-	16	10.8	148	3.24	0.98	0.64	1.66	NO	NO	-
67	414397	CHRONIC LIVER DISEASE	55	M	DM, COPD, CLD	16	10.8	168	3.24	0.98	0.72	1.9	NO	NO	-
68	1426847	ACUTE EXACERBATION COPD	44	F	-	16	11.4	168	2.58	0.58	0.34	1.08	NO	NO	-
69	1426927	DKA	66	M	DM	16	10.2	134	3.22	0.78	0.24	1.12	NO	NO	-
70	1426875	ACUTE POISONING	52	M	CAD	16	10.8	154	3.22	0.74	0.16	1.2	NO	NO	-
71	1199883	DKA	68	M	DM	14	8.6	100	2.54	0.96	0.34	1.8	YES	2	SACRAL
72	1437728	CHRONIC KIDNEY DISEASE	74	M	CKD HTN	16	8.6	112	1.45	0.9	0.34	2.1	NO	NO	-
73	1437014	CHRONIC KIDNEY DISEASE	70	F	-	16	8.9	166	2.58	0.92	0.34	1.8	NO	NO	-
74	1428641	DKA	42	F	DM	16	9.8	134	3.22	0.94	0.74	1.8	NO	NO	-
75	859279	CORONARY ARTERY DISEASE	60	M	DM,HTN	14	7.8	112	2.14	1.06	0.18	1.9	NO	NO	-
76	1437146	DKA	74	M	DM,CAD	18	8.8	130	3.08	0.96	0.24	1.9	NO	NO	-
77	1433783	SEPTICEMIA	86	M	DM	16	6.8	78	1.89	0.77	0.19	3.9	YES	2	SACRAL
78	1435995	UROSEPSIS	48	M	DM,HTN	18	8.8	128	2.88	0.78	0.64	1.09	NO	NO	-
79	1437067	ACUTE EXACERBATION COPD	55	F	COPD	18	9.6	122	1.43	0.77	0.24	1.01	NO	NO	-
80	755015	DKA	68	M	DM,CAD	16	10.8	156	2.99	0.67	0.16	3.5	NO	NO	-

Sr. No	MRD No	Diagnosis	Age	Sex	Coomorbidity	Braden scale	Hb	Platelets	Albumin	Direct bilirubin	Indirect bilirubin	Creatinine	Pressure sore	Stage of Pressure sore	Location
81	1410671	SEPTICEMIA	75	M	DM,COPD	18	9.6	178	2.5	1.68	1.24	1.9	NO	NO	-
82	1432076	METABOLIC SEIZURES	68	M	CKD HTN	-	6.2	98	2.04	2.8	1.66	2.9	YES	2	SACRAL
83	1437594	ISCHAEMIC STROKE	54	F	CKD HTN	16	10.4	166	3.45	2.8	1.98	2.04	NO	NO	-
84	623619	DKA	66	M	DM	14	8.2	102	2.7	1.68	0.77	3.1	YES	2	SACRAL
85	1437617	CHRONIC KIDNEY DISEASE	49	M	DM,HTN,CKD	16	7.4	68	2.01	3.09	2.66	4.9	YES	2	SACRAL
86	1436656	CHRONIC KIDNEY DISEASE	72	F	DM,COPD	14	6.8	110	1.88	2.08	1.99	3.9	YES	2	SACRAL
87	1438115	ACUTE FEBRILE ILLNESS	46	F	DM,HTN	14	13.2	144	2.8	1.99	0.44	1.4	YES	2	SACRAL
88	1212740	ISCHAEMIC STROKE	76	F	DM	16	7.2	109	2.1	2.9	1.89	3.8	YES	2	SACRAL
89	1433837	ACUTE EXACERBATION COPD	74	F	HTN	18	10.8	188	3.2	1.89	0.68	1.9	NO	NO	-
90	1249341	ACUTE EXACERBATION COPD	88	F	COPD	14	6.9	100	2.02	2.6	1.66	4.2	YES	2	SACRAL
91	1437769	ISCHAEMIC STROKE	78	M	-	16	8.2	115	2.45	2.56	1.64	3.1	YES	2	SACRAL
92	1433783	ACUTE FEBRILE ILLNESS	44	M	-	18	10.7	189	3.2	0.87	0.16	1.02	NO	NO	-
93	1436067	SEPTICEMIA	64	F	DM,HTN	16	11.4	156	2.4	1.08	0.68	0.89	NO	NO	-
94	1437140	CHRONIC KIDNEY DISEASE	72	M	DM	18	12.6	178	2.89	1.89	0.85	1.6	NO	NO	-
95	1435070	UROSEPSIS	52	F	DM	16	5.9	120	2.5	1.88	0.99	3.2	YES	2	SACRAL
96	1302684	METABOLIC SEIZURES	48	M	HTN	18	11.9	124	3.1	0.97	0.14	1.5	NO	NO	-
97	1406142	ACUTE EXACERBATION COPD	76	F	HTN	14	8	86	2.66	2.66	1.84	2.75	YES	2	SACRAL
98	1436012	UROSEPSIS	55	M	DM	18	8.5	102	1.67	1.08	0.23	1.2	NO	NO	-
99	1434580	CHRONIC KIDNEY DISEASE	68	F	DM,HTN,CKD	18	9.2	132	1.9	1.76	0.72	2.1	NO	NO	-
100	122065	DKA	50	F	DM	18	10.8	199	3.1	0.98	0.56	2.9	NO	NO	-
101	1337747	ACUTE EXACERBATION COPD	65	M	-	14	9.5	224	2.27	0.57	0.27	0.94	YES	2	SACRAL
102	1212740	SEPTICEMIA	68	F	DM,HTN	18	7.9	192	3.16	0.96	0.34	1.22	YES	2	SACRAL
103	1440306	UROSEPSIS	58	F	COPD	16	10.1	183	2.54	0.96	0.7	2.9	NO	NO	-
104	1437617	ACUTE EXACERBATION COPD	58	M	-	16	7.2	240	2.7	1.16	0.84	7.7	NO	NO	-
105	1198854	DKA	73	M	DM	16	9.7	332	2.3	1.7	0.8	1.11	NO	NO	-
106	1432076	UROSEPSIS	57	M	HTN	16	12.6	170	1.75	0.88	0.34	1.1	YES	2	SACRAL
107	1374655	SEPTICEMIA	43	M	CLD, ASCITES	12	7	120	1.6	0.8	0.26	6.66	YES	2	SACRAL
108	1437067	CORONARY ARTERY DISEASE	56	F	-	16	9.5	332	2.7	0.47	0.09	0.98	NO	NO	-
109	1438352	ACUTE EXACERBATION COPD	92	F	COPD	16	8.4	171	2.7	0.98	0.66	1	NO	NO	-
110	1439816	ACUTE TUBERCULAR MYELITIS	77	M	DM,HTN	16	11.4	108	2.73	2.48	0.79	0.34	YES	1	SACRAL
111	1415354	DKA	82	M	DM,HTN	16	8	128	2.14	0.34	0.11	6.85	YES	2	SACRAL
112	1790499	UROSEPSIS	66	M	DM,HTN	18	7.3	121	2.7	2.88	1.65	2.3	YES	2	SACRAL
113	1797418	ACUTE VIRAL HEPATITIS	47	M	HTN	18	9.6	114	3.56	9.8	6.56	1.9	YES	2	SACRAL
114	1226039	ACUTE EXACERBATION COPD	70	M	DM,COPD	18	6.8	98	2.12	1.88	0.79	3.9	NO	NO	-
115	1798994	ISCHAEMIC STROKE	77	M	HTN	18	10.7	144	2.89	0.89	0.16	1.89	NO	NO	-
116	1800963	ACUTE EXACERBATION COPD	56	M	HTN,COPD	18	11.8	167	2.8	0.45	0.16	1.5	NO	NO	-
117	1796733	METABOLIC SEIZURES	44	M	-	18	12.4	177	3.1	0.46	0.15	1.7	NO	NO	-
118	1798986	DKA	84	M	DM	16	9.2	102	2.11	0.89	0.14	2.9	NO	NO	-
119	1793703	CHRONIC KIDNEY DISEASE	47	M	CKD HTN	18	8.9	178	2.55	1.65	0.87	3.9	YES	1	SACRAL
120	1778430	HYPERTENSIVE EMERGENCY	50	M	HTN	16	10.7	115	2.1	1.49	0.83	2.7	YES	3	SACRAL
121	1801778	METABOLIC SEIZURES	58	M	HTN	18	8.8	98	1.89	2.78	1.66	3.7	YES	2	SACRAL

Sr. No	MRD No	Diagnosis	Age	Sex	Coomorbidity	Braden scale	Hb	Platelets	Albumin	Direct bilirubin	Indirect bilirubin	Creatinine	Pressure sore	Stage of Pressure sore	Location
122	1797988	CHRONIC KIDNEY DISEASE	74	M	CKD HTN	16	10.7	178	2.7	0.98	0.36	1.8	NO	NO	-
123	1798627	ACUTE VIRAL HEPATITIS	42	F	HTN	18	12.8	116	2.67	0.76	0.36	0.9	NO	NO	-
124	1784498	UROSEPSIS	58	M	DM	18	11.9	147	2.88	0.76	0.48	1.7	YES	2	SACRAL
125	1794221	ACUTE EXACERBATION COPD	62	M	COPD	16	7.8	67	2.1	3.88	2.16	4.1	NO	NO	-
126	1791507	ISCHAEMIC STROKE	52	M	DM	14	10.2	108	1.84	1.29	0.38	2.74	YES	1	SACRAL
127	1801558	TUBERCULAR MENINGITIS	57	F	DM,HTN	14	6.8	102	1.44	0.79	0.32	3.78	YES	2	SACRAL
128	1794259	DKA	64	M	DM	16	8.8	127	1.28	1.77	0.53	2.45	NO	NO	-
129	1801767	METABOLIC SEIZURES	38	M	HTN	18	10.6	224	2.89	0.99	0.42	1.78	NO	NO	-
130	1801117	GUILLAIN BARRE SYNDROME	72	M	DM,HTN	14	6.7	128	1.78	2.67	1.88	3.1	YES	UNSTAGEABL	SACRAL, B/L HEEL
131	1797387	ACUTE EXACERBATION COPD	49	M	COPD	16	12.7	178	1.89	0.75	0.12	1.9	NO	NO	-
132	1801744	UROSEPSIS	64	F	DM,HTN	14	8	111	1.8	0.78	0.16	3.69	YES	2	SACRAL
133	1777080	SEPTICEMIA	52	M	DM	18	13.4	121	2.8	?86	0.66	1.28	NO	NO	-
134	1781677	CORONARY ARTERY DISEASE	54	F	DM,HTN	18	10.5	159	2.1	1.56	0.74	1.67	NO	NO	-
135	1721194	ACUTE EXACERBATION COPD	69	M	COPD	16	9.6	125	2.89	0.66	0.12	1.06	NO	NO	-
136	1773372	DKA	50	M	DM	18	11.8	212	2.9	1.47	0.72	4.21	NO	NO	-
137	1780752	ISCHAEMIC STROKE	55	M	HTN	14	7.4	98	1.77	4.79	2.78	3.68	YES	3	SACRAL, OCCIPUT
138	1776979	ACUTE PANCREATITIS	46	M	-	18	13.9	168	3.2	1.67	0.73	1.76	NO	NO	-
139	1772640	ACUTE EXACERBATION COPD	68	M	HTN,COPD	18	12.6	189	2.78	0.89	0.23	2.1	NO	NO	-
140	1585843	CHRONIC KIDNEY DISEASE	59	F	DM,HTN	14	6.4	78	1.78	4.66	1.49	3.8	YES	UNSTAGEABL	SACRAL
141	1770769	SEPTICEMIA	68	F	CLD, ASCITES	14	8.2	178	2.55	1.99	0.52	4.8	YES	1	SACRAL
142	1775451	SEPTICEMIA	49	M	HCV	18	9.4	111	2.88	1.78	0.52	4.23	YES	3	SACRAL
143	1778426	LRTI	74	M	DM,COPD	14	8.6	199	1.78	2.69	1.22	2.8	NO	NO	-
144	1770036	ACUTE EXACERBATION COPD	69	M	COPD	16	10	221	2.75	1.89	0.47	1.2	NO	NO	-
145	1779215	UROSEPSIS	58	F	DM	18	7.9	108	2.58	0.76	0.44	2.11	YES	2	SACRAL
146	1781516	LRTI	74	M	HTN,COPD	16	10.6	167	2.48	1.58	0.62	1.98	NO	NO	-
147	1779118	LRTI	62	M	DM,COPD	18	9.8	227	2.9	1.9	0.62	1.11	NO	NO	-
148	1774788	SEPTICEMIA	50	M	DM,HTN,CKD	18	8.5	103	1.85	1.89	0.73	3.84	NO	NO	-
149	1778368	DKA	57	F	DM	16	11.4	115	2.49	0.78	0.16	1.69	NO	NO	-
150	1780987	SEPTICEMIA	39	F	-	18	9.2	185	2.99	1.25	0.18	2.67	NO	NO	-
151	1775247	METABOLIC SEIZURES	49	F	HTN	18	10.2	148	1.98	0.67	0.21	2.57	NO	NO	-
152	1778416	DKA	57	F	DM	16	9.46	184	2.64	1.84	0.61	3.14	YES	1	SACRAL
153	1772615	SEPTICEMIA	64	F	DM	18	6.9	28	1.26	5.78	2.95	6.8	YES	UNSTAGEABL	SACRAL
154	1775414	LRTI	72	M	COPD	14	10.2	111	2.75	1.72	0.17	2.11	NO	NO	-
155	147091	CHRONIC KIDNEY DISEASE	77	F	DM,HTN,CKD	16	7.7	99	1.11	2.16	1.52	3.8	YES	2	SACRAL
156	1776296	SEPTICEMIA	41	M	DM,HTN	18	10.2	111	2.71	1.72	0.81	2.88	NO	NO	-
157	1762435	DKA	57	F	DM,HTN	18	9.6	162	2.86	1.56	0.72	1.7	NO	NO	-
158	1776828	SEPTICEMIA	48	F	HIV	18	9.1	128	2.99	1.11	0.89	2.89	NO	NO	-
159	1770036	ACUTE FEBRILE ILLNESS	59	F	DM	18	11.5	185	2.71	0.92	0.17	3.26	YES	1	SACRAL
160	1775415	CORONARY ARTERY DISEASE	49	F	HTN	18	12.8	175	2.99	0.87	0.18	1.1	NO	NO	-
161	1773355	CHRONIC KIDNEY DISEASE	66	M	HTN,CKD	18	10	117	1.58	1.92	0.92	3.1	YES	2	SACRAL
162	1758975	LRTI	75	M	COPD	18	9.2	110	2.11	3.27	1.34	2.2	NO	NO	-

Sr. No	MRD No	Diagnosis	Age	Sex	Coomorbidity	Braden scale	Hb	Platelets	Albumin	Direct bilirubin	Indirect bilirubin	Creatinine	Pressure sore	Stage of Pressure sore	Location
163	1770719	SEPTICEMIA	60	M	DM	18	10.8	212	2.8	1.22	0.99	1.01	NO	NO	-
164	1754117	SEPTICEMIA	64	M	DM,HTN	18	11	199	2.5	0.87	0.12	0.89	NO	NO	-
165	1764053	ACUTE PANCREATITIS	51	M	DM	18	10	108	2.8	0.17	0.02	1.6	NO	NO	-
166	505313	DKA	73	M	DM,HTN,CKD	18	8.9	178	1.78	1.74	0.62	2.7	NO	NO	-
167	1764052	SEPTICEMIA	63	M	DM	18	11.2	128	1.79	0.95	0.32	1.88	NO	NO	-
168	1760155	DKA	66	F	DM,HTN	18	12.5	116	2.75	1.28	0.83	1.4	NO	NO	-
169	1727339	LRTI	78	M	DM,COPD	18	9.8	109	2.89	0.87	0.16	1.23	NO	NO	-
170	1757302	SEPTICEMIA	68	F	DM	18	8.1	104	1.89	2.89	1.24	3.1	NO	NO	-
171	1761481	DKA	70	M	DM	16	10.5	118	2.9	1.26	0.67	1.7	NO	NO	-
172	1756683	CHRONIC KIDNEY DISEASE	39	F	CKD HTN	16	117	2.4	1.68	0.75	4.8	1.28	NO	NO	-
173	1762679	GUILLAIN BARRE SYNDROME	52	F	HTN	18	162	2.89	1.1	0.83	0.22	1.1	NO	NO	-
174	1763987	SEPTICEMIA	66	M	DM,HTN,CKD	18	8	79	1.87	1.95	0.92	2.8	YES	2	SACRAL
175	1760192	ACUTE FEBRILE ILLNESS	40	M	-	18	11.9	179	3.8	0.98	0.24	0.89	NO	NO	-
176	177943	DKA	63	M	DM,HTN	18	9.9	124	2.9	1.27	0.64	1.12	NO	NO	-
177	1758975	UROSEPSIS	44	M	DM,HTN	18	8.1	101	3.5	1.88	0.85	2.79	NO	NO	-
178	1763384	CHRONIC KIDNEY DISEASE	39	F	CKD HTN	18	8.2	111	3.1	2.8	1.24	2.67	NO	NO	-
179	123776	ISCHAEMIC STROKE	69	F	DM,HTN	18	9.2	101	2.11	3.26	1.11	3.8	YES	3	SACRAL
180	1760651	LRTI	79	M	HTN,COPD	16	10.2	167	2.9	0.98	0.15	1.9	YES	2	SACRAL
181	1762483	SEPTICEMIA	68	M	DM,HTN	18	8.2	100	2.89	2.94	0.87	1.1	NO	NO	-
182	1761092	LRTI	56	F	DM	18	11.8	128	2.78	0.76	0.12	1.7	NO	NO	-

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Subject:: Approval of Plan of Thesis.

This is to inform you that the Faculty of Medical Sciences in its meeting held on 25.09.2023 has approved the plan of thesis of **Deepika Sharma**, a student of your college for **MCh Plastic & Reconstructive Surgery** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis
To evaluate the role of blood markers (haematological and biochemical) as predictors for development of pressure sores .

Supervisor
Dr. Ramneesh Garg
Professor
Dept. of Plastic & Reconstructive Surgery

Co-Supervisor:-
Dr. Rajinder K Mittal
Prof. & Head
Dept. of Plastic & Reconstructive Surgery

The student may submit the thesis in triplicate alongwith the plan in triplicate also, to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She may be directed to attach a photocopy of this letter with the forwarding letter while submitting thesis in the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic, if any.

Controller of Examinations

Note :

1. *If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.*

TO EVALUATE THE ROLE OF BLOOD MARKERS (HAEMATOLOGICAL AND BIOCHEMICAL) AS PREDICTORS FOR DEVELOPMENT OF PRESSURE SORES

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

M.Ch

IN

PLASTIC AND RECONSTRUCTIVE SURGERY

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES
FARIDKOT

OCTOBER, 2022

DR. DEEPIKA SHARMA

DEPARTMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY
DAYANAND MEDICAL COLLEGE & HOSPITAL
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ABSTRACT OF PLAN OF THESIS

Title	TO EVALUATE THE ROLE OF BLOOD MARKERS (HAEMATOLOGICAL AND BIOCHEMICAL) AS PREDICTORS FOR DEVELOPMENT OF PRESSURE SORES
For the degree of	M.Ch Plastic and Reconstructive Surgery
Name of the candidate	Dr. Deepika Sharma
Supervisor	Dr. Ramneesh Garg, Professor , Department of Plastic and Reconstructive Surgery , DMCH, Ludhiana
Co-supervisor	Dr. Rajinder K Mittal, Professor & Head, Department of Plastic and Reconstructive Surgery, DMCH, Ludhiana
Institution	Dayanand Medical College & Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: Pressure sores are an important public health problem associated with increased morbidity, reduced health- related quality of life, prolonged hospital length of stay, and high treatment costs. The Braden scale is one of the most widely used risk assessment scale for them with high negative predictive values. Present study is aimed to study haematological and biochemical parameters like haemoglobin, platelet count, serum albumin, direct and indirect bilirubin, and creatinine which can be considered for the risk evaluation for the pressure sore occurrence.

Aim of Study: To evaluate the role of blood markers (haematological and biochemical) as predictors for development of pressure sores.

Materials & Methods: This will be a prospective study in patients admitted to Medicine ICU whose Braden Assessment Scale will be done and patients with score less than 18 will be included in the study. Patients will be divided into two groups; Group A(patients without pressure sores and Group B (patients with pressure sores). Deranged value of recorded haematological and biochemical markers will be compared in both the groups using appropriate statistical methods.

Clinical Significance: To evaluate the role of blood markers (haematological and biochemical) in predicting pressure sore occurrence and comparing them with Braden Assessment scale.

Keywords: Braden Assessment Scale, Pressure Sore, Haematological and Biochemical Markers

(Signature) Candidate	(Signature) Co-Supervisor	(Signature) Supervisor	(Signature) Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"TO EVALUATE THE ROLE OF BLOOD MARKERS (HAEMATOLOGICAL AND BIOCHEMICAL) AS PREDICTORS FOR DEVELOPMENT OF PRESSURE SORES"** exist in the Department of Plastic and Reconstructive surgery, DMC&H, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis is genuine and is collected by the candidate herself under our supervision and guidance.

Supervisor

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Co-Supervisor

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Head of the Department

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Place : DMCH, Ldh

Date : 16/11/22

APPROVAL PROFORMA
BY RESEARCH & ETHICAL COMMITTEE
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

Name of candidate	Dr. Deepika Sharma
Department	Plastic and Reconstructive Surgery
Topic of Thesis	TO EVALUATE THE ROLE OF BLOOD MARKERS (HAEMATOLOGICAL AND BIOCHEMICAL) AS PREDICTORS FOR DEVELOPMENT OF PRESSURE SORES
Likely date of appearing in PG examination	May/June- 2024
Date of enrollment	28.12.2020
Name of Head of Department	Dr. Rajinder Mittal
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Dr. Harpreet Kaur (Professor & Head, Pathology)	Mr. R K Sharma (Legal Expert)
Dr. Jaspal Singh (Professor & Head, Surgery)	Dr. Ramit Mahajan (Associate Professor, Gastroenterology)
Dr. Dinesh Gupta (Professor & Head, Medicine)	Dr. Anu Sharma (Professor, Anatomy)
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Dr. Gurpreet S Wander (Vice Principal)	Dr. Gurpreet S Wander (Vice Principal & Professor & Head, Cardiology)
	Dr. Gagandeep Singh (Secretary, Ethics Committee Professor & Head, Neurology)

Approved: Yes / No

Dr. Sandeep Puri
Chairperson
Research Committee
Chairperson
Research Committee

Approved: Yes / No

Prof. Arvind Malhotra
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Ethics Committee
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Ethics Committee

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PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

1	NAME OF THE CANDIDATE AND ADDRESS	Dr. Deepika Sharma H. No 1489 Sector 32A urban estate Chandigarh road , Ludhiana
2	NAME OF THE INSTITUTE	DAYANAND MEDICAL COLLEGE AND HOSPITAL
3	COURSE OF STUDY AND SUBJECT	M.Ch Plastic and Reconstructive Surgery
4	DATE OF ADMISSION TO COURSE	29.04.2022
5	TITLE OF THE TOPIC	“TO EVALUATE THE ROLE OF BLOOD MARKERS (HAEMATOLOGICAL AND BIOCHEMICAL) AS PREDICTORS FOR DEVELOPMENT OF PRESSURE SORES”

6.0 BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR STUDY

Pressure sores are defined as soft-tissue injuries resulting from unrelieved pressure over a bony prominence. There are many causative factors for pressure ulcers like shear, friction, denervation, poor nutrition, age, dementia, moisture and smoking, other than the pressure which is the main causative factor.[1]

Pressure sore occurs in very ill patients and who are kept in prolonged immobilisation. They are quiet frequent in intensive care units and in paraplegic individuals. Due to the complicity of the long lasting treatment, the expenses for their care are high. Various scales are being used to predict pressure sore occurrence in an individual such as Braden Assessment Scale, NORTON Scale, Waterlow scale etc.

Braden Assessment Scale is the most commonly used scale for predicting pressure sore occurrence. It measures the risk for development of a pressure ulcer by using 6 subscales, each denoting a factor that has been found to contribute to pressure ulcer formation: mobility, activity, sensory perception, skin moisture, nutritional state, and friction/shear. Each of the subscales is scored from 1 to 4 (1–3 for friction/shear), with 1 representing the highest risk. The total Braden score ranges from 6 to 23. Lower total Braden score, higher is the risk of development of pressure sores. Eighteen is the cutoff score that is generally accepted in practice across clinical settings for predicting risk of pressure sores.[2] However, it showed low specificity and positive predictive value, which suggested a high false-positive rate. Overprediction may require unnecessary and costly interventions, which may not be a cost-effective use of health care resources. Therefore, a more accurate way of predicting which patients will have pressure sores develop in the ICU is needed.[3]

Many studies have been conducted on role of blood markers (haematological and biochemical) like haemoglobin, platelet count, serum albumin, direct and indirect bilirubin and creatinine in predicting pressure sores.[4]

The present study is designed to assess selected haematological and biochemical parameters of venous blood and their potential effects reflected by the development of pressure sores in patients treated in an intensive care setting.

6.2 REVIEW OF LITERATURE

Pressure sores are a type of injury that breaks down the skin and underlying tissue when an area of skin is placed under constant pressure for certain period causing tissue ischaemia, cessation of nutrition and oxygen supply to the tissues and eventually tissue necrosis. Constant pressure resulting in 'distortion or deformation damage' is probably the most accurate description of a pressure sore.

AETIOLOGY

There are many factors that can contribute to the development of pressure sores, but the final common pathway to ulceration is tissue ischaemia. The tissues are capable of sustaining pressure on the arterial side of around 30-32 mmHg for only a small duration of time. But when pressure increases even slightly above this capillary filling pressure, it causes microcirculatory occlusion and this in turn initiates ischaemia, tissue death and ulceration. Pressure sores can develop when a large amount of pressure is applied to an area of skin over a short period. They can also occur when less pressure is applied over a longer period. This can occur when a bony prominence is under persistent contact with an external surface. The most common site for pressure injuries is the sacrum. Other common sites include the heels, greater trochanter, ischial tuberosity, back of the head, ears, shoulders, elbows, inner knees, or malleoli. The majority of people affected with pressure sores are those having health conditions (mental or physical) that encourage immobility, especially those who are confined to bed or chair for prolonged periods of time.[5]

There are various extrinsic and intrinsic factors responsible for development of pressure sores. Extrinsic factors are pressure, shear, friction, immobility and abnormal posture. Intrinsic factors responsible for pressure sore development are altered consciousness, decreased or altered sensations, nutritional deficiencies, anemia, edema etc.[6,7]

Pressure effect is directly proportional to the body weight and the duration for which it is applied and is inversely proportional to the surface area of the skin in contact. Constant pressure for long period induces ischemia and causes reactive hyperemia. Muscles and subcutaneous tissues are more susceptible to pressure induced injury than the skin, therefore pressure ulcers are generally worse than what they appear on the skin surface.[8] Apart from the direct effect of pressure causing ischemic tissue necrosis, the reperfusion after relieving the pressure leading to inflammatory tissue destruction by macrophages also participates in the causation of ulceration.[9]

Shearing is forceful inter-tissue plane movement which causes stretching and tearing of blood vessels, reduced blood flow, stasis and ischemic tissue necrosis and it usually occurs when a semi-reclining patient glides on the bed or on the operation table because of gravity or when the patient is turned and pushed on the bed without lifting him/her off the surface.[10]

Friction between the skin and the stationary, surfaces, such as bed clothes results in the loss of stratum corneum. The extrinsic moisture from perspiration, urine, feces and discharge causes maceration of the skin surface and forms blisters. Sensorimotor feedback system is impaired in neurologically compromised patients and in those who have received prolonged anesthesia or in sedated patients due to which their body fails to make postural adjustments in response to prolonged pressure and ischemia.

Neurological conditions causing paralysis or motor weakness prevents change of posture in patients when pressure is exerted. Hemoglobin is a

good indicator of the patient's nutritional status. Good hemoglobin is required for tissue oxygenation. In anemic patients, oxygen carrying capacity of blood is reduced and hence, there is decreased supply of oxygen to the tissues which precipitates tissue necrosis in ischemic tissue because of mechanical pressure.[11]

GRADES OF PRESSURE SORES:

The most widely accepted classification system is the National Pressure Ulcer Advisory Panel (NPUAP) system.[12] It uses ulcer depth as a way to classify these ulcers.

The stages are as follows:

- Stage I: The skin is intact with the presence of non-blanchable erythema.
- Stage II: There is partial-thickness skin loss involving the epidermis and dermis.
- Stage III: There is a full-thickness loss of skin that extends to the subcutaneous tissue but does not cross the fascia beneath it.
- Stage IV: There is full-thickness skin loss extending through the fascia with considerable tissue loss. There might be possible involvement of the muscle, bone, tendon, or joint.

ASSESSMENT OF PRESSURE SORE:

Pressure sores are a significant financial burden to healthcare systems. A recent systematic review noted that the cost for prevention of pressure ulcers was lower than that for treatment.

Use of pressure sore risk assessment tools or scales is a component of the assessment process to identify individuals at risk of developing a

pressure ulcer. Risk assessments generally use checklists that alerts to the most common risk factors that predispose individuals to pressure sore development. These checklists are often developed into risk assessment tools, for example the Norton Scale, the Waterlow Scale and the Braden Scale.[13]

Braden Assessment Scale is most frequently used scale for predicting pressure sore occurrence. It was developed in 1984 by Barbara J. Braden and was published in 1987.[14-15] The Braden scale evaluates the risk of skin breakdown in six domains: sensory perception, moisture, activity, mobility, nutrition, and friction and shear.

It is given below:

BRADEN SCALE – For Predicting Pressure Sore Risk

SEVERE RISK: Total score ≤ 9 **HIGH RISK**: Total score 10-12 **MODERATE RISK**: Total score 13-14 **MILD RISK**: Total score 15-18				DATE OF ASSESS ➡				
RISK FACTOR	**SCORE/DESCRIPTION**				**1**	**2**	**3**	**4**
SENSORY PERCEPTION Ability to respond meaningfully to pressure-related discomfort	**1. COMPLETELY LIMITED** – Unresponsive (does not moan, flinch, or grasp) to painful stimuli, due to diminished level of consciousness or sedation, **OR** limited ability to feel pain over most of body surface.	**2. VERY LIMITED** – Responds only to painful stimuli. Cannot communicate discomfort except by moaning or restlessness, **OR** has a sensory impairment which limits the ability to feel pain or discomfort over ½ of body.	**3. SLIGHTLY LIMITED** – Responds to verbal commands but cannot always communicate discomfort or need to be turned, **OR** has some sensory impairment which limits ability to feel pain or discomfort in 1 or 2 extremities.	4. **NO IMPAIRMENT** – Responds to verbal commands. Has no sensory deficit which would limit ability to feel or voice pain or discomfort.				
MOISTURE Degree to which skin is exposed to moisture	1. **CONSTANTLY MOIST**– Skin is kept moist almost constantly by perspiration, urine, etc. Dampness is detected every time patient is moved or turned.	2. **OFTEN MOIST** – Skin is often but not always moist. Linen must be changed at least once a shift.	3. **OCCASIONALLY MOIST** – Skin is occasionally moist, requiring an extra linen change approximately once a day.	4. **RARELY MOIST** – Skin is usually dry; linen only requires changing at routine intervals.				
ACTIVITY Degree of physical activity	1. **BEDFAST** – Confined to bed.	2. **CHAIRFAST** – Ability to walk severely limited or nonexistent. Cannot bear own weight and/or must be assisted into chair or wheelchair.	3. **WALKS OCCASIONALLY** – Walks occasionally during day, but for very short distances, with or without assistance. Spends majority of each shift in bed or chair.	4. **WALKS FREQUENTLY**– Walks outside the room at least twice a day and inside room at least once every 2 hours during waking hours.				
MOBILITY Ability to change and control body position	1. **COMPLETELY IMMOBILE** – Does not make even slight changes in body or extremity position without assistance.	2. **VERY LIMITED** – Makes occasional slight changes in body or extremity position but unable to make frequent or significant changes independently.	3. **SLIGHTLY LIMITED** – Makes frequent though slight changes in body or extremity position independently.	4. **NO LIMITATIONS** – Makes major and frequent changes in position without assistance.				
NUTRITION Usual food intake pattern [1]NPO: Nothing by mouth. [2]IV: Intravenously. [3]TPN: Total parenteral nutrition.	**1. VERY POOR** – Never eats a complete meal. Rarely eats more than 1/3 of any food offered. Eats 2 servings or less of protein (meat or dairy products) per day. Takes fluids poorly. Does not take a liquid dietary supplement, **OR** is NPO[1] and/or maintained on clear liquids or IV[2] for more than 5 days.	**2. PROBABLY INADEQUATE** – Rarely eats a complete meal and generally eats only about ½ of any food offered. Protein intake includes only 3 servings of meat or dairy products per day. Occasionally will take a dietary supplement **OR** receives less than optimum amount of liquid diet or tube feeding.	**3. ADEQUATE** – Eats over half of most meals. Eats a total of 4 servings of protein (meat, dairy products) each day. Occasionally refuses a meal, but will usually take a supplement if offered, **OR** is on a tube feeding or TPN[3] regimen, which probably meets most of nutritional needs.	4. **EXCELLENT** – Eats most of every meal. Never refuses a meal. Usually eats a total of 4 or more servings of meat and dairy products. Occasionally eats between meals. Does not require supplementation.				
FRICTION AND SHEAR	1. **PROBLEM**- Requires moderate to maximum assistance in moving. Complete lifting without sliding against sheets is impossible. Frequently slides down in bed or chair, requiring frequent repositioning with maximum assistance. Spasticity, contractures, or agitation leads to almost constant friction.	2. **POTENTIAL PROBLEM**– Moves feebly or requires minimum assistance. During a move, skin probably slides to some extent against sheets, chair, restraints, or other devices. Maintains relatively good position in chair or bed most of the time but occasionally slides down.	3. **NO APPARENT PROBLEM** – Moves in bed and in chair independently and has sufficient muscle strength to lift up completely during move. Maintains good position in bed or chair at all times.					
TOTAL SCORE	**Total score of 12 or less represents HIGH RISK**							

ASSESS	DATE	EVALUATOR SIGNATURE/TITLE	ASSESS.	DATE	EVALUATOR SIGNATURE/TITLE
1	/ /		3	/ /	
2	/ /		4	/ /	

Each subscale is ranked on a scale of 1 to 4, except for the friction/shear subscale, which is scored in a scale of 1 to 3. Subscores are added to determine the cumulative score that varies from 6 to 23; lower scores indicate higher risk for pressure ulcer development. A cutoff score of 18 or less indicates an increased risk for pressure ulcer development.[16]

Several studies have shown that the Braden Scale demonstrates reasonable accuracy for predicting pressure sore development, and reported sensitivity ranging from 43.5% to 89.0% and specificity measurements from 43.5% to 83.1%. In a study by Schoonhoven and colleagues, the Braden Scale showed a positive predictive value of 8.1% and a negative predictive value of 94.9%.[17-18]

In addition to the above, there are other haematological and biochemical parameters like haemoglobin, platelet count, serum albumin, direct and indirect bilirubin, and creatinine that can be considered for the risk evaluation for the development of pressure injury. It is known that low levels of hemoglobin and platelets are related to the pathological condition of anemia, which involves a lower transport of nutrients and oxygen to the tissues, and they are responsible for breaking skin integrity.[19]

Many studies have been conducted to predict the role of biochemical markers like albumin, direct and indirect bilirubin and creatinine in prediction of pressure sores. The present study is aimed to evaluate the role of various haematological and biochemical markers in development of pressure sores and its comparison with Braden Assessment Scale.

6.3 AIMS AND OBJECTIVES-

- To evaluate the role of blood markers (haematological and biochemical) as predictors for development of pressure sores.

7.0 MATERIAL AND METHODS:

7.1 SOURCE OF DATA:

This prospective study will be conducted on patients admitted in Medicine ICU at Dayanand Medical College and Hospital, Ludhiana from October 2022 to February 2024.

7.2 METHOD OF COLLECTION OF DATA

INCLUSION CRITERIA-

- Patients admitted to Medicine ICU.
- Age more than 18 years.
- Braden scale less than 18.

EXCLUSION CRITERIA-

- Patients with pre-existing pressure sore.
- Patients not willing to participate in the study.
- ICU stay of less than 5 days.

7.3 Study design

- This will be a prospective study on patients being admitted to Medicine ICU.
- Informed consent will be taken from the patient/family before including the patient in the present study.
- All the patients will undergo detailed assessment within 12 hours of admission in ICU in the form of history and clinical examination of the skin over all bony prominences.
- Braden Assessment Scale will be used daily to identify the patients who are at risk for pressure ulcer development.

BRADEN SCALE – For Predicting Pressure Sore Risk

SEVERE RISK: Total score ≤ 9 **HIGH RISK**: Total score 10-12
MODERATE RISK: Total score 13-14 **MILD RISK**: Total score 15-18

RISK FACTOR	SCORE/DESCRIPTION				DATE OF ASSESS ➡ 1	2	3	4
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FRICTION AND SHEAR	1. **PROBLEM**- Requires moderate to maximum assistance in moving. Complete lifting without sliding against sheets is impossible. Frequently slides down in bed or chair, requiring frequent repositioning with maximum assistance. Spasticity, contractures, or agitation leads to almost constant friction.	2. **POTENTIAL PROBLEM**– Moves feebly or requires minimum assistance. During a move, skin probably slides to some extent against sheets, chair, restraints, or other devices. Maintains relatively good position in chair or bed most of the time but occasionally slides down.	3. **NO APPARENT PROBLEM** – Moves in bed and in chair independently and has sufficient muscle strength to lift up completely during move. Maintains good position in bed or chair at all times.					
TOTAL SCORE	**Total score of 12 or less represents HIGH RISK**							

ASSESS	DATE	EVALUATOR SIGNATURE/TITLE	ASSESS.	DATE	EVALUATOR SIGNATURE/TITLE
1	/ /		3	/ /	
2	/ /		4	/ /	

NAME-Last	First	Middle	Attending Physician	Record No.	Room/Bed

Form 3166P BRIGGS, Des Moines, IA 50306 (800) 247-2343 www.BriggsCorp.com
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BRADEN SCALE

Use the form only for the approved purpose. Any use of the form in publications (other than internal policy manuals and training material) or for profit-making ventures requires additional permission and/or negotiation.

- Patients with Braden scale less than 18 will be included in this study.
- Haematological and biochemical parameters like haemoglobin, platelet count, serum albumin, Direct and indirect bilirubin, and creatinine will be recorded for these patients **biweekly** until their discharge.
- Head-to-toe skin examination over bony prominences will be done to document the presence and stage of pressure sore.
- Patients will be divided into two groups:-
  Group A -patients without pressure sores.
  Group B- patients with pressure sores.
- Derranged value of recorded haematological and biochemical markers will be compared in both the groups using appropriate statistical methods.

DURATION OF STUDY: October 2022 to February 2024.

OUTCOME: To compare the role of Braden Assessment Scale and Various haematological and biochemical markers as efficient predictors of pressure sore occurrence.

STATISTICAL ANALYSIS: Data will be described in terms of range; mean ±standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. To determine whether the data is normally distributed, a Kolmogorov-Smirnov test will be used. Comparison of quantitative variables between the study groups will be done using Student t-test and Mann Whitney U test for independent samples for parametric and non-parametric data respectively. For comparing categorical data, Chi square ($\chi 2$) test will be performed and fisher exact test will be used when the expected frequency is less than 5. A probability value (p value) less than 0.05 will be considered statistically significant. All statistical calculations will be done using (Statistical

Package for the Social Science) SPSS 21version (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

Yes, Haemoglobin, platelet count, serum albumin, direct and indirect bilirubin, and creatinine.

7.4 HAS ETHICAL CLEARENCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Attached herewith

8. LIST OF REFERENCES

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PROFORMA

- NAME- AGE/SEX-
- CR No.- Admission No.-
- DIAGNOSIS-
- DATE OF DISCHARGE-
- COMORBIDITIES-
- PAST HISTORY-
- BRADEN SCALE-

	Day1	Day2	Day3	Day4	Day5	Day6	Day7	Day8	Day9	Day10
Braden Scale										

- Haematological Markers-

	Day1	Day4	Day7	Day10	Day13	Day16	Day19
Haemoglobin							
Platelet count							

- Biochemical Markers-

	Day1	Day4	Day7	Day10	Day13	Day16	Day19
Albumin							
Direct bilirubin							
Indirect bilirubin							
Creatinine							

- DEVELOPMENT OF BED SORE-
- STAGE OF PRESSURE SORE-
- (DATE AND DURATION OF HOSPITAL STAY) CONDITION ON DISCHARGE-

Signature of candidate **Signature of Supervisor**

- Picture of pressure sore:

Signature of candidate **Signature of Supervisor**

INFORMED PATIENT CONSENT

Date: _____

I……..D/o, W/o, S/o **"TO EVALUATE THE ROLE OF BLOOD MARKERS (HAEMATOLOGICAL AND BIOCHEMICAL) AS PREDICTORS FOR DEVELOPMENT OF PRESSURE SORES"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

……. ……

Signature of the Witness Signature of the patient

Name:

Signature of Candidate **Signature of Supervisor**

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____

निवासी _____

इस शोधकार्य **"TO EVALUATE THE ROLE OF BLOOD MARKERS (HAEMATOLOGICAL AND BIOCHEMICAL) AS PREDICTORS FOR DEVELOPMENT OF PRESSURE SORES"** में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____ _____

गवाह के हस्ताक्षर रोगी के हस्ताक्षर/अंगूठे का निशान

नाम: नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____

“TO EVALUATE THE ROLE OF BLOOD MARKERS (HAEMATOLOGICAL AND BIOCHEMICAL) AS PREDICTORS FOR DEVELOPMENT OF PRESSURE SORES” ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

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ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____ ਮਿਤੀ_____

ਪਤਾ _____ ਪਤਾ _____

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ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

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ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ